# HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED SKIN LESIONS

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*Dedicated*

*to*

*my family*

*&*

*Teachers*

# *ACKNOWLEDGEMENT*

*"As we express our gratitude, we must never forget that the highest appreciation is not to utter words, but to live by them".*

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*DR. PURNIMA*

# LIST OF ABBREVIATIONS

| | |
|---|---|
| H&E | Haematoxylin and eosin |
| FDE | Fixed Drug Eruption |
| LTR | Lichenoid tissue reaction |
| IFD | Interface dermatitis |
| HPV | Human Papilloma virus |
| HLA | Human Leucocyte Antigen |
| HSV2 | Herpes Simplex Virus 2 |
| HCV | Hepatitis C virus |
| HIV | Human immunodeficiency virus |
| LP | Lichen Planus |
| LPSA | Lichen planus specific antigen |
| UVA | Ultraviolet A |
| PIH | Post inflammatory hyperpigmentation |
| CDKN2A | Cyclin dependent kinase inhibitor 2A |
| CDK4 | Cyclin dependent kinase 4 |
| MC1R | Melanocortin 1 receptor |
| BRAF | B-Raf proto-oncogene serine/threonine kinase |
| BCC | Basal Cell carcinoma |
| DNA | Deoxyribonucleic acid |
| WHO | World Health Organisation |
| ET-1 | Endothelin 1 |
| PVI | Perivascular inflammation |
| PLCA | Primary localized cutaneous amyloidosis |
| EN | Erythema nodosum |
| BD | Bowen’s Disease |
| SCC | Squamous cell carcinoma |
| DLE | Discoid lupus erythematosus |
| PIH | Postinflammatory hyperpigmentation |

# ABSTRACT

**Introduction**: All the hyperpigmented skin lesion biopsies will be stained with H&E stain after fixation in 10% formalin and analysed in detail. Special stains and IHC will also be performed wherever required.

**Aim of Study**: To analyse the histological spectrum of hyperpigmented skin lesions.

**Materials & methods**: Data for the prospective study will be obtained from all the skin biopsies of hyperpigmented skin lesions received in the Department of Pathology, DMCH, over a period of 1.5 years. All the slides will be stained with H&E stain after fixation in 10% formalin and analysed in detail. Special stains and IHC will also be performed wherever required. These findings will be correlated with clinical profiles of the patients.

**Results:** Majority of the hyperpigmented skin lesions presented in the third decade with female preponderance. All the lesions were classified into inflammatory lesions, benign and malignant tumors. Classical lichen planus was the most common lesion followed by lichen simplex chronicus and lichen planopilaris. Melanocytic nevi were the most common presenting lesions among the benign tumors and basal cell carcinoma was the most common among the malignant tumors. Of the 90 hyperpigmented cases, 63 cases (70%) showed histopathological correlation with the clinical diagnosis while 27 cases (30%) were diagnosed only on histopathological examination.

**Conclusion:** Hyperpigmented lesions of the skin can have a variable etiology ranging from infectious diseases to malignant tumors. The exact diagnosis can only be achieved by a thorough correlation between the pathologist and the dermatologist.



**Keywords**: hyperpigmentation, benign, malignant, lichen planus, melasma

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# INTRODUCTION

At 15% of the total weight of the human body, the skin is regarded as the largest organ in the body. It generates a protective layer, has functions according to its role, and, most importantly, pledges to preserve normal homeostasis via its barrier function.[1] In India, skin disorders are among the most common health issues, with a prevalence ranging from 6.3% to 11.6%.[2] The pigment known as melanin is responsible for the wide spectrum of different hues and gradations of human skin tone.[1]

Skin conditions can manifest in a variety of ways, ranging from benign vesicular lesions to hyperpigmented lesions and deadly malignant tumors. Many skin conditions include non-specific, infectious, and non-malignant diseases as well as a variety of neoplasms, including both benign and malignant tumors. The range of dermatological problems is wide and differs between countries and even within the same country. Numerous factors, including sex, age, and any accompanying systemic ailments, as well as socioeconomic considerations, literacy levels, social standards, and racial traits, ensure this variation.[3]

Although there are many different types of skin disorders, most are first diagnosed based on a clinical history and visual examination. When there are questions about the diagnosis, a skin biopsy is a crucial tool for confirmation. Since it is only carried out in a limited number of instances, a profitable yield is always preferred. This is dependent on a number of variables, including the biopsy site, sufficient sample size, biopsy technique, and relevant clinical and physical results. Clinico-pathological discordance occurs when there is a significant difference between the clinical diagnosis and the histological findings.[4]

Problems with pigmentation are thought to be a significant contributing factor to dermatological consultations. Significant skin tone difference has a negative psychological effect on an individual's quality of life and mental health in India. Skin lesions with varying levels of pigmentation can result from the amount of melanin in human skin.[1] Increased skin and mucous membrane pigmentation is referred to as

hyperpigmentary skin lesions. An anomaly in the dermis or epidermis may lead to hyperpigmentation. There are two categories for hyperpigmentary skin disorders: "blue hyperpigmentation" or "Ceruloderma" refers to changes in the dermis, and "brown hyperpigmentation" refers to abnormalities in the epidermis.[5] The majority of dermatopathologists treat pigmentary skin disorders, which include a wide range of histological traits from benign tumors like nevi to malignant tumors like malignant melanoma.

Different etiologies can lead to pigmentary disorders.

• Aberrant melanocyte migration from the neural crest to the skin during development

• Compromised melanosome transfer to the epidermal keratinocytes

• Altered the process of melanin synthesis.

• Melanocytic destruction induced by poisons or immunological mechanisms

• A variety of medications, such as diuretics and antibiotics, stimulate melanogenesis and raise the amount of melanin in big melanocytes.

• Tyrosinase enzyme activity is inhibited by heavy metal-induced inactivation of sulfhydryl compounds in the skin, leading to skin hyperpigmentation. Melanogenesis increases when this inhibition is removed.[6]

Ephelides (freckles), melasma, solar lentigines, post-inflammatory hyperpigmentation, and café-au-lait macules are among the common causes of hyperpigmentation in the body. Even though the majority of hyperpigmented lesions are benign and the diagnosis is simple, it's crucial to rule out melanoma and its precursor lesions as well as recognize the systemic disease's skin symptoms. Treatment options for solar lentigines, ephelides, melasma, and post-inflammatory hyperpigmentation include topical medications, chemical peels, laser therapy, and cryotherapy. While the majority of pigmentation problems are benign or nonspecific, some offer significant cosmetic and psychological challenges for the patient, necessitating assessment and intervention. Others can be signs of a primary skin cancer or an

underlying systemic illness. A correct diagnosis of these prevalent skin disorders will enable the doctor to help the patient receive the right skin care and to reassure them. [7]

Hyperpigmentation can indicate underlying organ malfunction in addition to being a cosmetic abnormality that deforms people and causes psychological distress.

In order to begin appropriate treatment when a patient seeks medical attention for skin lesions, the doctor needs a precise diagnosis. Nonetheless, because skin lesion appearance and clinical data always overlap, the clinical diagnosis of skin diseases can be difficult. When skin biopsies are not histopathologically examined, accurate diagnosis is frequently lacking.[8]

A pathologic examination is frequently used to supplement or validate a diagnosis. A consistent treatment regimen for hyperpigmented skin lesions may be established with the aid of the histology-based therapeutic principles. Literature on the clinico-histopathological investigation of hyperpigmentary skin lesions in India is scarce. It is commonly recognized that skin lesions evolve into distinct stages as the disease advances and that clinocopathologic correlation is necessary for the histologic diagnosis of inflammatory and other skin diseases. The clinical diagnosis of skin diseases might be difficult on the basis of clinical history and the emergence of skin lesions. In the absence of a histopathologic analysis of skin samples, there may be insufficient data to support an accurate diagnosis.[9]

# AIMS AND OBJECTIVES

To analyse the histological spectrum of hyperpigmented skin lesions.

# REVIEW OF LITERATURE

There is a diverse range of people with various skin tones on our globe. The skin was once thought to be a protective layer covering more sensitive and complex interior viscera, more than a century ago. The skin was then regarded as essentially a passive barrier to prevent fluid loss and mechanical harm throughout the next century. Numerous studies on melanocytes, clinical features, and the management of pigmentary illnesses have been conducted recently.Around the world, there are regional variations in the prevalence of pigmentary disorders.[6]

Three layers make up skin: the epidermis,the dermisand the subcutaneous adipose tissue. Skin functions are incredibly varied.

- It regulates body temperature and electrolyte levels
- It acts as a mechanical barrier against physical, chemical, and biologically harmful substances
- It is an immune organ
- It is a crucial organ of sensuality and psychological health.

Furthermore, it is a medium through which internal organ disorders as well as primary skin diseases are expressed.

**EMBRYOLOGY**

Ectodermal cells form a single layer that gives rise to the epidermis. This separates into two layers by the fifth week of gestational age, at least focally: the stratum germinativum, or basal layer, and the periderm that covers it.

- The stratum intermedium, an intermediary layer, forms by 10 weeks.
- At 19 weeks, the periderm starts to flatten and there are multiple layers of intermediate cells.[10]

## SKIN'S NORMAL STRUCTURE

### *EPIDERMIS*

Through the process of desquamation, the epidermis, a stratified and keratinizing squamous epithelium, dynamically renews itself while retaining its typical thickness. The epidermis's cells comprise of:[10,11]

(a) Keratinocytes

(b) Melanocytes

(c) Langerhans cells

(d) Toker cells (in certain anatomic locations)

(e) Merkel cells

Moreover, hair follicles and eccrine ducts (acrosyringium) have apertures in the epidermis. According to recent immunohistochemistry research, Langerhans cells and free nerve axons are present in the epidermis.

By 23 weeks, keratinization is fully established in the stratum intermedium along with tiny granules of keratohyaline. The newly created stratum corneum is represented by the keratinizing cells that remain beneath the majority of the lost periderm cells at this point.

### *Keratinocytes*

From bottom to top, the keratinocytes of the epidermis are arranged into four ordered layers: [10,12,13]

(a) Basal layer (stratum basale, germinativum)

(b) Squamous layer (stratum spinosum, prickle cell layer)

(c) Granular layer (stratum granulosum)

(d) Cornified or horny layer (stratum corneum)

***1. Basal layer***: The mitotically active cells known as basal cells are the progenitors of other keratinocytes. From a histological perspective, basal cells are viewed as a single layer of cells that are situated above

the basement membrane. Their size, shape, and melanin concentration vary slightly. Columnar or cuboidal in shape, basal cells have a basophilic cytoplasm.

Round or oval in shape, the nucleus has an inconspicuous nucleolus and granular chromatin. Melanin is present in the cytoplasm of basal cells due to pigment transfer from nearby melanocytes. Specialized areas in the plasma cell membranes called desmosomes link basal cells to keratinocytes and to each other. They are joined to the subepidermal basement membrane via hemidesmosomes, which are modified desmosomes, and are oriented perpendicular to it. Keratin intermediate is present in basal cells and the squamous cells that cover them.

***2. Squamous layer:*** The squamous layers consist of five to ten layered cells, with bigger keratinocytes than basal cells. The cytoplasm of the polyhedral suprabasal keratinocytes is slightly basophilic, and the nucleus is spherical. Many of these keratinocytes have sporadic patches of melanin, which acts as a shield against UV radiation damage.

Larger, flatter, eosinophilic, and orientated parallel to the surface characterize the more superficial cells. Within the cytoplasm of the keratinocytes are one or two prominent nucleoli and tono filaments. Due to the distinctive appearance of small projections stretching from cell to cell seen under light microscopy, the squamous layer is also known as the spinous or prickle cell layer.

***3. Granular layer***: One to three layers of flattened cells arranged parallel to the skin's surface make up the granular layer. Keratohyalin granules are highly basophilic-stained granules found in the cytoplasm. Hematoxylin and eosin (H&E)-stained sections, on the other hand, show red staining of trichohyalin granules, which are generated by the inner root sheath of hair follicles. The protein filaggrin, which encourages keratin filament aggregation in the cornified layer, is derived from the histidine-rich keratohyalin granules.

***4. Cornified layer***: Multiple layers of polyhedral eosinophilic keratinocytes devoid of cytoplasmic organelles and a nucleus make up the cornified layer. These are the keratinization system's highest differentiated cells. All of the high molecular weight keratin filaments that make them up are present. The cornified layers are placed in a basket-weave pattern in the formalin-fixed area. Eventually, these cells will fall off the skin'ssurface.

Keratinization takes 20 to 45 days to complete. The lowest part of the cornified layer, above the granular layer, contains a homogenous eosinophilic zone called the stratum lucidum in histologic sections obtained from the skin of the palms and soles. Rich in extracellular components like active enzymes and SH groups, this extra layer adds to the skin.

**Langerhans cells**

Paul Langerhans identified Langerhans cells (LCs) in 1868. These are dendritic, mobile, and antigen-presenting cells that are found in all stratified epithelium, but are most common in the mid to the uppermost squamous layer sections. At high magnification, LCs can be inferred in H&E-stained sections because they appear to lie within lacunae with darkly stained nuclei that have an indented, reniform appearance. Similar to melanocytes, routine slices are unable to reveal their dendritic character. Histoenzymatic stains for adenosine triphosphatase (ATPase) can be used to identify Langerhans cells; alternatively, immunoreactivity for S-100 protein and, more precisely, the antibody to the CD1a antigen can be used to identify them in formalin-fixed, paraffin-embedded tissue. Histoenzymatic and immunohistochemical stains show that LCs are extensively dendritic.[10]

***Merkel cells***

Merkel cells (MCs), which were initially identified by F.S. Merkel in 1875, are dispersed and asymmetrically distributed in the epidermis's basal cell layer. They might form clusters together. Together, these expanded terminal sensory nerve fibers constitute

progressively adapting mechanoreceptors, which convey touch feeling within the epidermis. They are more concentrated in the tactile hair disks, the outer root sheath of hair follicles, and the glabrous skin of the lips, digits, and oral cavity. Regular histologic preparations fail to identify Merkel cells. To identify them, immunostaining and electron microscopy are needed. Desmosomes allow MCs to bind to nearby keratinocytes under electron microscopy.

Their nuclei are invaginated, their cytoplasms are sparse, and their paranuclear array of cytokeratin filaments is parallel and the distinctive dense core granules that are membrane-bound and frequently, though not always, associated with unmyelinated neurites.[10,14]

**Toker cells**

It's crucial to understand that occasionally, cells in the squamous layer will have transparent or pale cytoplasm. It's important to identify these cells from the cancerous ones that cause Paget's disease.

**Melanocytes**

Dendritic cells derived from the neural crest are called melanocytes. Melanocytes from the neural crest may migrate and settle in different epithelia. The melanocytes are found in the basal layer of the epidermis, and their dendritic processes reach in every direction. Melanocytes in H&E preparations consist of ovoid or elongated nuclei encircled by a transparent cavity.[16,17] Generally speaking, they are smaller than the nearby basal keratinocytes. Melanocytes do not bind to basal cells via desmosomes and do not have tono filaments. Laminin-5, a constituent of anchoring filaments that stretch from the melanocytes' plasma membrane to the basal lamina, could function as a ligand to facilitate the attachment of melanocytes to the basement membrane in vivo. Furthermore, melanocytes at the basal lamina have characteristics that resemble hemidesmosomes of keratinocytes.[18]

**Formation of Melanin**

The intricate process of melanin manufacturing is seen in melanocytes, which are highly specialized cells that include membrane-bound organelles known as melanosomes. Melanosomes are transported to nearby keratinocytes by dendrites, where they are essential for photoprotection.[19,20] Each melanocyte is thought to be in touch with keratinocytes in the basal and suprabasal layers. This physical link between keratinocytes and melanocytes is known as "the epidermal melanin unit." Melanosomes, orgenells related to lysosomes, are where melanin is synthesized. Melanosomes are membrane-limited vesicles found in the Golgi-associated endoplasmic reticulum during the early stages of development. There are four phases in melanosome maturation: [16,20,21]

Stages I through IV have ellipsoidal melanosomes with many longitudinal filaments

- Stage I melanosomes are spherical and devoid of melanin.
- Melanin deposits begin at stage II and become noticeable in stage III.
- Stage IV melanosomes are fully packed, with melanin concealing the interior structures.

The neighboring basal keratinocytes and hair follicular cells receive the developing melanosomes along with their melanin content.

***DERMIS***

Adnexal structures, vascular and nerve plexuses, and cells, fibrous tissue, and ground substances are all present in the dermis, which is a dynamic and supporting connective tissue. There are two zones in the dermis:[12,14,15]

- Reticular dermis
- Papillary dermis

The papillary and periadnexal dermis are combined to form the adventitial dermis. A delicate branching network of small elastic fibers and a loose meshwork of thin, poorly organized collagen, mostly type

III collagen mixed with some type I collagen, are characteristics of the papillary and periadnexal dermis. A delicate branching network of small elastic fibers and a loose meshwork of thin, poorly organized collagen, mostly type III collagen mixed with some type I collagen, are characteristics of the papillary and periadnexal dermis.

***SUBCUTANEOUS TISSUE***

The subcutaneous tissue, often referred to as the hypodermis or subcutis, is essential for energy production, heat regulation, insulation, and defense against mechanical trauma. It is made up of lobules made of mature adipose tissue. The spherical, cytoplasmically lipid-rich adult adipocytes inside the lobules compress the nucleus against the side of the cell membrane. Vimentin and S-100 protein are expressed by adipocytes in immunohistochemical staining. The interlobular septum, which is made up of thin bands of dermal connective tissue, divides these adult adipocyte lobules.

Skin pigmentation is a prevalent ailment that can be initiated by a multitude of circumstances. Genetics, sun exposure, and specific drugs are the three main factors that contribute to skin pigmentation. It will be easier to treat and prevent skin pigmentation if we are aware of its basic causes.

It may be possible to anticipate an individual's melanocyte count through genetics. Skin cells called melanocytes are responsible for producing melanin. Nevertheless, when tanning and hyperpigmentation occur, the melanin-containing organelles called melanosomes need to be moved and grown, but they also shrink during hypopigmentation. People with darker skin tones are more likely to have higher quantities of melanin, the pigment that gives skin its colour.

Skin pigmentation is frequently caused by sun exposure. The body creates more melanin to protect itself from the sun's UV radiation. To protect the skin from the sun's rays, this could make the skin more pigmented.

Several medications may also lighten the skin’s pigment. One class of drugs, antibiotics, can boost melanin synthesis, increasing skin colour. When certain medications, such as birth control pills, are taken together, skin pigmentation may also intensify. A person taking medicine should speak with their doctor to find out if the medication could impact the colour of their skin.

A person's skin might get darker (hyperpigmentation) or lighter (hypopigmentation) when they are unhealthy. Low melanin concentration, also known as hypopigmentation, is most frequently caused by prior skin trauma, which includes skin sores like blisters, infections, burns, chemical exposure, and other wounds. The skin will appear paler than the surrounding skin surface once an injury has healed.

Sun exposure is the most common cause of hyperpigmentation since it greatly increases the synthesis of melanin. Prolonged light exposure could exacerbate dark patches by making them bear similarities to aging spots, post-inflammatory patches, and melasma. Melasma and chloasma are two instances of hyperpigmentation caused by hormonal reasons. This condition is common in women and has been linked to the female sex hormones progesterone and oestrogen, which increase the production of melanin when the body is exposed to sunlight. It is also seen following treatment with hormone replacements. As we age, the number of melanocytes decreases, but the ones that remain develop and become more specialized. These physiological changes demonstrate how aging manifests itself more visibly in those over 40 years of age. Pigmentation is influenced by genetics.[9]

Since hyperpigmentary skin illnesses are a set of extremely heterogenic diseases ranging from non-neoplastic infectious diseases to neoplastic benign and malignant tumors, any attempt to classify them is far from satisfactory.
Some of the common hyperpigmentary skin disorders are discussed below.

**Ephelids**

Freckles, also known as ephelids, are tiny pigmented macules that mainly appear on the face but are less common in other sun-exposed locations. Freckles are sporadic, 1-2 mm tiny macules that are typically found on the nose and malar region.

Histological characteristics include no increase in melanocyte count and hypermelanization of the basal epidermis.[4]

**Melasma**

Melasma, a frequent acquired condition characterized by symmetric hyperpigmentation, is more common in women and people with darker skin tones. It usually affects the face. Its pathophysiology has been linked to a number of etiological factors, including familial history, sun exposure, and hormone effects. Melasma histological features include bigger melanocytes, increased melanosomes, dermal blood vessels, and sporadic perivascular lymphohistiocytic infiltrates. It can also show enhanced epidermaland/or dermal pigmentation.[22]

Treatments for melasma that include topical, oral, and procedural approaches have been effective.

A retrospective study by Muhammad Ali et alexamined patients who complained of skin hyperpigmentation between January and December of 2016. Melasma accounted for 33.63% of the skin darkening in 7124 patients, while post-inflammatory hyperpigmentation was responsible for 12.52% of cases. Consequently, coming to a conclusion that melasma was the leading cause of hyperpigmentation.[23]

**Fixed drug eruption**

Following each exposure to the offending medication, it manifests as recurrent plaques at fixed locations such the lips, vaginal areas, and acral sites. These plaques don't hurt, and post-inflammatory hyperpigmentation is how they naturally go away.

Histologically, the dermaoepidermal junction is obscured by necrotic keratinocytes and lymphocytic infiltrates. One notable characteristic is

pigment incontinence. Dermal melanophages might be the sole indicator of a fully healed FDE lesion.[24]

**Ochronosis**

Exogenous ochronosis, a blue-black discoloration on the skin, is caused by using hydroquinone-containing bleaching products. In terms of clinical involvement, pigmentation is related to bony prominences on the forehead, temple, nose, lower jaw, and side of the neck. Histopathology reveals basophilic and enlarged collagen fibers. Homogentisic acid, a yellow-brown ochronotic pigment, coats collagen fibers in older lesions, giving them the appearance of banana-shaped bodies. Neither Masson's fontana nor Perl's stain will cause the pigment to discolor. The histopathological characteristics of exogenous and endogenous ochronosis are similar. [25]

**Poikiloderma of civatte**

The majority of cases of poikiloderma of civatte are observed in middle-aged, postmenopausal Caucasian women, while people with darker skin tones have also been observed. Skin lesions affect the parts of the face, neck, and chest that are exposed to the sun. In a clinical setting, hyperpigmentation is observed as reticulate patches.[26]

Histologically, it is distinguished by a pronounced solar elastosis along with fibrosis, melanophages, and telengectasia in the papillary dermis. The accumulation of elastotic material can be more clearly seen when stained with Orcein.[27]

**Cutaneous amyloidosis**

Since it resembled starch or cellulose, Virchow was persuaded to adopt the word "amyloid" in 1854. In 1842, Rokintansky provided the initial account of amyloidosis. Localized or systemic amyloidosis is possible. The term "primary localized cutaneous amyloidosis" (PLCA) refers to skin-specific amyloidosis that lacks systemic involvement.In addition, skin treated with UVA radiation following psoralens intake,

seborrheic and actinic keratosis, Bowen's disease, and porokeratosis all exhibit secondary cutaneous amyloid deposits.

Three main categories can be used to categorize PCLA:

1) Macular amyloidosis

2) Papular lichen amyloidosis

3) Amyloidosis nodule (tumefactive)

The predominant areas of dark brown, rippling pigmentation associated with macular amyloidosis are the upper back, arms, and forearms. The hallmark of lichen amyloidosis is a persistent, itchy, papular lesion that primarily affects the lower limbs. Nodular amyloidosis is an uncommon form of Primary Cutaneous Amyloidosis that typically affects the face or legs and is characterized by one or more 1-3 cm nodules. It is unclear how cutaneous amyloidosis etiopathogenesis occurs. Although the etiology of PCLA appears to be complex, genetic and environmental factors seem to be major players in its pathophysiology. The continuous friction caused by pumice stones, coconut fibers, and nylon bath sponges is one of the suggested reasons for keratinocyte degeneration.The disease known as amyloidois is caused by the extracellular deposition of fibrillar proteins, which damages tissue and impairs function. The accumulation of amyloid in what seems to be normal skin is known as primary localized cutaneous amyloidosis.[10]

Amyloid is visible histopathologically as eosinophilic material stained with H&E. The diagnosis is verified by apple green birefringence under polarized microscopy in conjunction with Congo red staining. According to Venugopal SB et al study, the pretibial region was most affected by the bulk of pruritic lesions associated with cutaneous amyloidosis. Under a microscope, the most consistent findings across all instances were hyperkeratosis, amyloid deposits in the papillary dermis, and perivascular inflammation.[28]

**Riehl’s melanosis**

A reaction to cosmetics can cause pigmented contact dermatitis, which manifests as grey-brown pigmentation. It mostly affects women in their middle years. The illness is a Type IV hypersensitivity reaction that

causes pigment incontinence, basal cell vacuolization, and perivascular or band-like dermal infiltrates in the initial lesion. Diffuse, patchy, or reticular patterns with satellite, perifollicular pigmented macules are possible.[29]

**Idiopathic eruptive macular pigmentation**

Asymptomatic brown macular lesions covering the neck, proximal limbs, and trunk are the initial signs of the condition. Children and teenagers are most frequently affected by this illness. Histology reveals epidermal basal layer pigmentation without degeneration, and the lesions might go away in a few months.[30]

**Seborrhoeic keratosis**

One of the most prevalent benign pigmented epidermal tumors is seborrheic keratosis. They typically start to develop without any sex predeliction after the age of fifty.[30,31]

Risk factors include human papilloma virus (HPV), sunshine, and heredity.

These are tan to brown or black papules that are somewhat elevated. Lesions can appear anywhere on the skin, with the exception of the palms and soles, although sun-exposed skin is more frequently afflicted. They are typically easily removed and have a stuck-on appearance.

Epidermal keratinocytes proliferate either endophytically, exophytically, or flatly in seborrheic keratoses. The histologic appearances of seborrheic keratosis differ.[30]

Acanthosis, papillomatosis, and hyperkeratosis are present in all forms of seborrheic keratosis. The acanthotic epidermis typically has two types of cells: basaloid and squamous. The former resemble squamous cells, which are typically seen in the epidermis; in contrast, basaloid cells are tiny, homogeneous, and have a sizable nucleus.[32] Because malignant tumors can grow elsewhere on the body or, very rarely, inside a seborrheic keratosis, follow-up is crucial for individuals

with numerous seborrheic keratoses.Unless the growths become irritating or unsightly, treatment is typically not necessary. Growths may be surgically removed or removed by cryotherapy (freezing) if treatment is required.[30]

**Lichenoid eruption**

In dermatology and pathology, lichenoid tissue reaction and interface dermatitis are two of the most frequently occurring clinical and histological manifestations.

The discovery of an inflammatory infiltration in a skin biopsy that obstructs the dermo-epidermal junction is referred to as interface dermatitis.

Histopathologically, focal parakeratosis and minor basal vacuolar alterations with a few eosionophils and plasma cells are characteristics that distinguish lichenoid eruptions from one another.[33,34]

**Lichen planus**

Lichen planus, a hyperpigmented skin disease that affects the skin, mucous membranes, hair follicles, and nails, is the most often documented type of lichenoid tissue reaction in the general population.[34,35]

It's an eruption with violaceous, flat-topped papules that are typically itchy, however the cause is uncertain. Pickham's striae, a network of tiny white lines, can be observed on the surface of papules. The flexor surfaces of the genitalia, thighs, trunk, and wrists are preferred.[10] Although the pathophysiology of lichen planus is unknown, various explanations have been put forth. These include genetic, allergic, psychosomatic, immunological, and infectious. Lichen planus has been linked to a number of autoimmune conditions, including lichen sclerosus, ulcerative colitis, diabetes mellitus, and dermatomyositis. Genetically, HLA-A3, HLA-A5, and HLA-B7 are linked to a higher incidence of lichen planus. Happenings and contact sensitizers might be involved in the initiation of lichen planus.[35]

Immunologically, cytotoxic CD8+T cells, which make up the majority of infiltrates in the dermo-epidermal interface, are responsible for the basal cell degeneration. The Langerhans cells present the novel antigens that have been processed against the injured keratinocytes to the helper T cells. According to Shiohara and Mizukawa's 2005 description, the main pathogenic event in lichen planus is cell-mediated immunity. Chronic bladder infections, amoebiasis, syphilis, HSV2, HCV, and HIV have all been linked to lichen planus.[35]

Lichenoid interface dermatitis is a characteristic of LP. A dense, continuous, and band-like lymphohistiocytic infiltrate in the upper dermis and at the dermal-epidermal junction are among the characteristic histological findings. LP lesions present with a variety of epidermal alterations, such as squamatization, compact hyperkeratosis or orthokeratosis, focal areas of wedge-shaped hypergranulosis, basilar vacuolar degeneration, and minor spongiosis in the spinous layer.[35]

There are often dome-shaped dermal papillae between the elongated rete ridges. Necrotic keratinocytes are seen at the dermalepidermal junction and in the basal layer of the epidermis. "Colloid or civatte bodies" are eosinophilic remains of anucleate apoptotic basal cells that can also be seen in the dermis. Whickham striae are typically observed in hypergranulosis-affected areas.

It is possible to observe vacuolar degeneration at the basal layer, which results in isolated subepidermal clefts (Max Joseph spaces). A genetically predisposed person's altered epidermal cells are a result of their cell-mediated immune response to an induced antigen, which could be a medication, a contact allergen, a virus, or another infectious agent. Lichen planus specific antigen (LPSA), an MHC Class I antigen, is recognized by CD8+ infiltrates in the lesional skin. About 80% of patients have it, and it is unique to lichen planus. Olsen et al. used autologous lesional skin in 1983 to demonstrate LPSA utilizing an indirect immunofluorescence method. It was lacking in the dermis and foundation membrane zone, although it was present in the strata granulosum and spinosum.[35,36]

**Variants of Lichen Planus**[30,35]

- **Hypertrophic Lichen planus**: These lesions typically last for several years and manifest as one or more pruritic plaques that may seem verrucous. Rarely, long-standing lesions will progress into squamous cell carcinoma. In this variation, keratoacanthoma, verrucous cancer, and cutaneous horns are also possible outcomes.

  Histopathology reveals hypergranulosis, pronounced irregular acanthosis, and orthokeratosis in the epidermis. At the base of the rete ridges, the vacuolar change and the lymphocytic inflammatory infiltrate are particularly noticeable.
- **Atrophic Lichen planus**: Lichen planus papules are typically found at the borders. The epidermis is thin and the typical rete ridge pattern has disappeared, according to histology. Compared to a regular lichen planus, the infiltrate is typically less dense.
- **Lichen planus actinicus*:***Lesions are restricted to body parts that are exposed to sunlight, making it a unique clinical variation of lichen planus. With a trend towards epidermal thinning at the lesion center and more noticeable pigmentary incontinence in the upper dermis, the histologic appearance is comparable to that of normal lichen planus. Not all cases of lichen planus exhibit an infiltration of inflammatory cells as densely as those that do.
- **Lichen planopilaris (follicular lichen planus**): This is a clinically diverse form of lichen planus where keratotic follicular lesions coexist with other lichen planus symptoms. The most prevalent and significant clinical category is distinguished by scalp baldness with scarring. Histopathology reveals a lichenoid response pattern involving the follicular epithelium's basal layer, along with a high perifollicular lymphocyte infiltrate and a sparse histiocyte population. The follicle's isthmus and infundibulum both undergo modifications.

- **Lichen striatus*:*** It is an etiologically unknown linear, papular eruption that typically extends the length of an extremity on one side of the body, either continuously or intermittently. Patients with darker skin tones may exhibit hypopigmented lesions. Histopathology reveals a lichenoid reaction pattern, in which three or four nearby dermal papillae are occupied by lymphocytes, histiocytes, and melanophages.
- **Lichen nitidus*:***It is an uncommon, typically asymptomatic chronic eruption that is characterized by numerous, tiny, flesh-colored papules that measure one or two millimeters in diameter. Histopathology reveals a well-circumscribed mixed-cell granulomatous infiltration limited to enlarged dermal papillae. It is made up of mononuclear and a few multinucleated epithelioid histiocytes, as well as lymphocytes. Vacuolar change of the basal cell layer, focal parakeratosis, decreased granular layer, and focal subepidermal clefting are also observed. The material appears to be grasped by the rete ridges, which have a tendency to bend downward and inward like a "claw clutching a ball."
- **Drug induced Lichen planus**: There have been reports of lichenoid eruptions after ingesting a variety of chemicals and medications. Common medicines that cause lichenoid eruptions include gold, methyldopa, β-adrenergic blocking agents, penicillamine, quinine, quinidine, synthetic antimalarials, and ethambutol.

  The presence of focal parakeratosis, minor basal vacuolar alteration, a few eosinophils, and occasionally plasma cells in the infiltrate characterize lichenoid drug eruptions, setting them apart from lichen planus. Compared to the lichen planus itself, the infiltrate is frequently less bandlike and dense. Compared to normal lichen planus, civatte bodies can be observed above the basal layer and have greater melanin incontinence.

In their study, Mruthunjayappa et al demonstrated that classical lichen planus and its variants accounted for the majority of biopsy-confirmed hyperpigmented lesions, with a rate of 55%. According to histopathology, interface dermatitis was present in the majority of the lesions.[30]

According to Gourohi F. et al, vulvar lichen planus can affect a person's physical and mental health in addition to interfering with sexual activity. Infection with HPV and HCV are also more common in these patients.[35]

**Prurigo nodularis**

A persistent dermatitis known as prurigo nodularis is typified by distinct, elevated, hard, hyperkeratotic papulonodules that range in size from 5 to 12 mm and are extremely itchy. Their predisposition is towards the extensor surfaces of the limbs.

The cause of prurigo nodularis is a mystery. Numerous illnesses, ranging from psychiatric disorders to systemic or metabolic ailments, have been documented to cause prurigo nodularis.

Prolonged mechanical damage to the skin, such as scratching, causes pigmentary changes, an increase in dermal nerve fibers, plaques or nodular lichenification, and hyperkeratosis.

Notable hyperkeratosis and uneven acanthosis are observed on histology. The typical neurological alteration observed in these patients is cutaneous nerve hypertrophy and proliferation.[37]

**Morphea**

Morphea is distinguished by its mauve or violet hue with a lilac border. Increased melanocytic activity is visible on Fontana Masson stained slices as noticeable melanocytes in the basal cell layer. Melanin incontinence and basal cell degeneration are absent. Morphea's histopathology is divided into three stages: early inflammatory, middle, and late sclerotic.

The first lesion exhibits somewhat thicker collagen bundles surrounded by interstitial lymphoplasmacytic infiltrates, either with or without eosinophils.

In the late sclerotic stage, the inflammatory infiltrate completely disappears and there are more thicker collagen bundles with nearly normal epidermis.[38]

**Post inflammatory hyperpigmentation**

Post-inflammatory hyperpigmentation is a result of sun damage, especially in those with darker skin tones. Examples of sun damage include acne lesions, ingrown hairs, scratches, and insect bites. It may affect the dermis or the epidermis and manifests as distinct, hyperpigmented macules with fuzzy feathery borders. Histologically, the skin that has been exposed to the sun has more inflammatory cells. The basal cell layer of the epidermis has more melanin pigment in epidermal postinflammatory hyperpigmentation.

The upper dermis is affected by dermal postinflammatory hyperpigmentation, and pigment incontinence is brought on by an increase in melanophages in the papillary dermis.[39]

In their study, Krishma Goyal et al. demonstrated that lichen planus pigmentosis and post-inflammatory hyperpigmentation were the two main causes of pigmentary diseases.[40]

From a histopathological perspective, every instance exhibited an infiltration of perivascular lymphocytic inflammatory cells, and most of them displayed pigment incontinence. Increased basal cell pigmentation, hyperkeratosis, and basal cell degeneration are further characteristics of PIH.[39]

**Lichen simplex chronicus**

This skin condition is represented by a thicker, centrally lichenified plaque that is frequently hyperpigmented due to persistent scratching.

The neck, ankles, scalp, vulva, pubis, scrotum, and extensor forearms are the most frequently affected areas.[41]

The most common site of involvement is the scrotum in men and the labia majora in women. There is a higher incidence of anogenital lichen simplex chronicus in men. The age range of 35 to 50 years old is the greatest occurrence, with women being more impacted than men.[21] The primary characteristic used to distinguish cases of lichen simplex chronicus was the lichenified plaque with papules and excoriation. Epidermal hyperplasia, orthokeratosis, and hypergranulosis with regular rete ridge elongation are the histological characteristics of lichen simplex. Periodically, macrophages and lymphocytes penetrate the perivascular space. Many people choose capsaicin, topical glucocorticoid creams like betamethasone dipropionate, intralesional corticosteroids like triamcinolone, antipruritics, and topical glucocorticoids as recommended treatments for lichen simplex chronicus.[41]

**Hyperpigmentation due to physical agents**

Physical agents may promote hyperpigmentation by exposing skin to UV radiation, with UVA radiation being the most prevalent type that tans the skin. Other factors include heat radiation, ionizing radiation, and trauma.[30]

**Nutritional deficiency disorders**

Numerous cutaneous symptoms are known to be caused by nutritional deficient illnesses. Among these include malabsorption syndrome and vagabond's disease; however, vitamin deficiencies (vitamins A, B3, B12, folic acid, and C) are the most often occurring conditions.[30]

**Endocrinopathies**

They may result in a range of cutaneous symptoms, such as skin darkening, which frequently indicates an underlying endocrine disorder. These alterations may be brought on by endocrinopathies, pregnancy, or oral contraceptives.[30]

**Melanocytic lesions**

The architecture and cytomorphological appearance of composite cells within melanocytic lesions display a significant degree of morphological variability.

Studies have revealed a few characteristics that can predict the development of melanocytic nevi, the most significant of which is childhood sun exposure. Strong genetic regulation governs the formation of nevi, while environmental exposures impact the average number of nevi.

Melanocytic proliferations are made up of one or more of three related cell types that can be seen in the epidermis, dermis, or infrequently the subcutis: melanocytes, nevus cells, and melanoma cells. Melanocytes may proliferate locally, resulting in benign or malignant melanocytic neoplasms, which are defined by melanocytes arranged in close proximity to one another to create a tumor.[11]

Melanocytic nevi are the common term for benign melanocyte tumors, whereas malignant melanomas are the term for tumors of the melanocytes; the cells that make up these lesions are referred to as nevus cells and melanoma cells, respectively.[42]

**Melanocytic nevi**

The widespread consensus is that melanocyte proliferation in melanocytic nevus is a benign neoplastic growth. Rarely do melanocytotic nevi exist at birth. Most nevi first show up in early adulthood and youth.

Nevi go through transitional phases in their life cycle; it is thought that they begin as junctional nevi, develop into compound nevi, become intradermal nevi, and finally undergo involution.[43]

**Junctional Nevi**

It appears as a brown or black macule that can appear anywhere on the body and is most commonly noticed in early adolescence or youth. It is made up of well-defined nests of nevus cells that are either

completely contained inside the lower epidermis or protrude into the dermis while remaining in contact with the epidermis. Normally uniform and cuboidal in shape, the nevus cells in these nests are sometimes spindle-shaped. The nevus cells have varying numbers of melanin granules.[10]

**Compound Nevi**

They affect kids and teenagers more frequently. In clinical terms, a compound nevus is a plaque or papule that is pigmented. The characteristics of an intradermal and junctional nevus are combined in a compound nevus.

Nevus cells may exhibit distinctive morphologic differences known as types A, B, and C, correspondingly, in the upper, middle, and lower dermis.

Type A nevus cells are typically round to cuboidal in shape and exhibit a large amount of cytoplasm that contains different amounts of melanin granules in the upper dermis. Occasionally, melanophages might be observed in the surrounding stroma. Type B cells are typically seen in the mid-dermis. Compared to type A cells, they are smaller, have less cytoplasm and melanin, and typically form distinct cords or clusters. Because type C nevus cells in the lower dermis are often elongated and have a spindle-shaped nucleus, they often mimic fibroblasts or Schwann cells. Seldom do they include melanin, and they frequently lie in strands.[10]

**Intradermal Nevus**

The bulk of these melanocytic nevi, which are the most prevalent kind, are discovered in adults.

Nevus intra dermal exhibits almost minimal junctional activity. Nevus cells are found only in the dermis, where they form ropes and nests. You might notice multinucleate cells. Nevius cells in the deeper layers of the lesion may take on a neuroid appearance.

When treating a benign melanocytic nevus, the primary goal shifts to cosmetic enhancement. Numerous methods of treatment, both surgical and non-surgical, have been documented. Before the age of ten, there is a significant risk of cancer associated with large congenital melanocytic nevi. Consequently, early and thorough excision followed by reconstruction is the current suggested course of treatment for these lesions. As a novel form of therapy, laser treatment has advanced remarkably in recent years.[10,11]

**Malignant melanoma**

Hippocrates coined the phrase "black cancer" in the fifth century BC to refer to cutaneous malignant melanoma. The term "melanoma" was first used in a published work by Rene Laennec in 1806, who also gave the first description of the condition.

The sixth decade of life is when the incidence peaks.[44] The head and neck region and lower extremities are where they are most frequently found, with the latter being more prevalent in females. Nowadays, it is generally acknowledged that ultraviolet radiations, which are further classified as ultraviolet A (UVA), ultraviolet B (UVB), and ultraviolet C (UVC), are the main environmental risk factor for the development of primary cutaneous melanoma. Genetic susceptibilities (such as CDKN2A, CDK4, MC1R, BRAF, and p16/ARF) and environmental exposure (UV light) cause melanocytes to accumulate genetic alterations that activate oncogenes, inactivate tumour suppressor genes, and impede DNA repair processes. Melanocyte proliferation, blood vessel expansion, tumor invasion, immune response inhibition, and finally metastasis result from this. Histology reveals a highly noticeable perivascular distribution in the dermis coupled with the presence of both extracellular and intracellular melanin.[45]

Melanoma can develop inside an already-existing benign melanocytic nevus or it can happen de novo, meaning it doesn't have a precursor lesion. Melanoma can develop from any form of melanocytic nevi,

however some are more likely to do so than others, such as congenital melanocytic nevi.

Malignant melanoma tumor thickness and lymph node metastases are thought to be significant prognostic variables. [46,47]

Melanoma can develop inside an already-existing benign melanocytic nevus or it can happen de novo, meaning it doesn't have a precursor lesion. Melanoma can develop from any form of melanocytic nevi, however some are more likely to do so than others, such as congenital melanocytic nevi. It has long been known that there are various subtypes of melanoma, and that these subtypes differ in their etiology and place of occurrence. The melanoma classification that Clark and colleagues developed more than 30 years ago, which uses the body site of the primary melanoma and morphologic features of the early (radial) growth phase to separate four basic categories, is the basis for the current WHO classification.[47]

1. Superficial spreading melanoma (SSM)
2. Lentigo maligna melanoma (LMM)
3. Nodular melanoma (NM)
4. Acral lentiginous melanoma (ALM)

The idea of radial and vertical growth phases in the development of malignant melanoma was first presented by Clark and associates. Clark's degree of infiltration: has both descriptive and predictive value.[48]

Level 1- confined to dermis
Level 2- Invasion into papillary dermis
Level 3- Invasion into papillary and reticular dermis interface
Level 4- Invasion into reticular dermis
Level 5- Invasion into subcutaneous fat

According to a study by Nam KM et al., the lower extremities are the most frequently affected areas for primary skin malignancies, followed by the feet, upper extremities, trunk, head, and neck. Acral lentiginous melanoma was the most often observed histological subtype, followed

by nodular melanoma, superficial spreading melanoma, and lentigo malignant melanoma.[49]

**Nevus of ota and ito**

The sole differences between Nevus of Ota and Ito, two benign melanocytic disorders, are the sites of involvement and dissemination. The distribution of the trigeminal nerve is affected by Nevus of Ota, primarily the ophthalmic V1 and maxillary V2 divisions. It also manifests as hyperpigmentation of the eyes, hence its alternate term, ocular dermal melanosis.

Nevus of Ito affects the posterior supraclavicular nerves and the lateral cutaneous brachial nerves of the shoulder. With nevus of ito, ocular problems are not observed.

The pathophysiology of these two disorders is identical, with more melanocytes in the dermis. In their work, Agarwal P et al. demonstrated that there is no conclusive diagnostic test for either of these disorders. The primary methods for diagnosing them are clinical examination and history. A skin biopsy is required if there is pigmentation variation or an ulceration of the skin layer above.[50]

**Progressive systemic sclerosis**

Multiple physical signs of systemic sclerosis, a chronic multisystem connective tissue condition, are common.

It is typified by microangiopathy, which causes fibrosis and inflammation in the skin and underlying organs. It is unknown what precise mechanism causes the body's diffuse coloring.
In their research, Viswanath V et al. identified the melanocytic potential of endothelin-1 (ET-1), a substance generated by keratinocytes that promotes melanogenesis by stimulating the multiplication of melanocytes and the creation of melanin, as the cause of the body's hyperpigmentation.[51]

**Urticaria pigmentosa**

It is sometimes referred to as mastocytosis and is characterized by an increase of mast cells in the lymphatic, liver, spleen, bone marrow, and GIT.

In the later stages of mastocytosis, diffuse cutaneous involvement is observed. It is thought to be a benign, self-resolving illness that goes away in puberty.

Urticaria pigmentosa can be diagnosed clinically, but a skin sample is necessary for a definitive diagnosis. Mast cell increase in the dermis is urticaria pigmentosa's signature finding.

In their work, Macri et al. demonstrated that there was no increase in mast cells in the dermis in biopsy specimens taken from the patient with mastocytosis who had normal skin. In certain cases, a GIT or bone marrow biopsy may be required.[52]

**Granuloma annulare**

The most prevalent non-infectious granulomatous illness of unknown cause on the skin is Granuloma annulare (GA). It is usually self-limiting and benign. On the upper extremities, erythematous plaques or papules organized in an annular pattern are typically the initial signs of granuloma annulare. Apart from the typical manifestation known as localized granuloma annulare, there are additional distinct clinical variations of this condition, including generalized, perforating, patch, and subcutaneous forms. Even though it's a benign disease, it can be linked to more terrifying conditions like HIV or cancer. There is no known etiology or pathophysiology for GA. According to some research, a delayed-type hypersensitivity reaction—more precisely, a Th1 reaction involving IFN-gamma activating macrophages to release matrix metalloproteinases—causes granuloma annulare. The end product of this entire process is the breakdown of connective tissue. [53]

According to certain research' findings, a compromised neutrophil response causes macrophages to gain control of an inflammatory location, resulting in the granulomatous inflammation associated with granuloma annulare rather than a suppurative neutrophil-type

inflammation.

The histopathological features of granuloma annulare include mucus accumulation, inflammation with interstitial histiocytes, and localized collagen degradation. It is possible to distinguish between four distinct histological patterns: mixed, palisaded granulomatous, sarcoidal granulomatous, and interstitial. A rim of lymphocytes and histiocytes encircles a region of necrobiotic collagen in the palisading pattern. Histiocytes strewn among collagen and blood vessel bundles in the dermis form the interstitial pattern. A patient could fit into more than one histopathologic subtype category.[54]

**Erythema nodosum**

Erythema nodosum (EN) is characterized by the acute emergence of firm, solid, deep nodules or plaques that are erythematous in colour. It is a common acute nodular septal panniculitis. These lesions are primarily found on the extensor surfaces of the legs and are palpably painful. Regardless of the cause, these nodules have a common histological appearance. It is characterized by sudden inflammation of the hypodermic fat's interlobular septa and dermo-hypodermic junction without any necrosis or after effects.[55]

A nonspecific cutaneous reaction to different antigens causes EN. Immunological mediation is the pathophysiological mechanism for EN. The idea of a type IV delayed hypersensitivity response to several antigens is supported by a wealth of direct and indirect evidence. The creation of granulomas, the generation of oxygen free radicals, the deposition of immune complexes in the venules of subcutaneous fat, and TNF-alpha are all part of the pathogenesis.

Skin biopsies are not very helpful in diagnosing common types. A biopsy is recommended when there are nodules that have atrophic scarring, fistulisation, atypical topography, or nodules that persist for several weeks. It emphasizes how a lymphohistiocytic inflammatory infiltrate involves interlobular septa while sparing fat lobules and arteries.[56]

**Lupus erythematosus**

The multisystem condition lupus erythematosus primarily affects the skin. Cutaneous lupus comes in various forms. Acute cutaneous lupus (ACLE), subacute cutaneous lupus (SCLE), and discoid lupus (DLE) are the three most prevalent kinds. DLE is the most prevalent kind or variation of chronic cutaneous lupus erythematosus. Lesions are often photo distributed and tend to have subsequent atrophy or scarring; these individuals may or may not experience photosensitivity. Significant systemic illness is not present in the majority of DLE patients. About 20% of patients may also experience DLE as a symptom of SLE. The etiology of cutaneous lupus erythematosus is complex, involving the interaction of both hereditary and environmental variables. Among the environmental factors that contribute are ultraviolet radiation (UVR), drugs, tobacco use, and potentially viruses.

An inflammatory cascade involving cytokine, chemokine, and inflammatory cell responses is brought on by the interplay of these several variables. TYK2, IRF5, and CTLA4 genes have been linked to SLE in the past and enhance the likelihood of developing DLE.[57]

**Bowen's disease**

JT Bowen originally described in situ squamous cell carcinoma (SCC) in 1912, and this condition is known as Bowen's disease (BD). Clinically speaking, a typical case of BD is a well-demarcated, slowly growing erythematous patch or plaque with a crusted or scaling surface. It may occasionally be verrucous or pigmented. It usually appears on the head, neck, and lower limbs. However, palmar, vaginal, perianal, subungual, and periungual BD are also observed.[58]

According to Pedersen et al. (1988) and Cox et al. (2007), 10% to 20% of cases of BD involve numerous sites, while most cases are solitary lesions. For extragenital lesions, the chance of progression into an invasive carcinoma is 3% to 5%, and for genital lesions, it is approximately 10% (Kao 1986; Cox et al 1999). A number of etiological factors have been linked to bilateral breast cancer (BD), including ultraviolet irradiation, radiotherapy, photochemotherapy, carcinogens

(such as arsenic), immunosuppression (such as after organ transplantation, AIDS), viruses (there is a strong correlation between HPV and genital and perianal lesions; the HPV genome is present in 47% of acral and 24% of nonacral extragenital BD), and other conditions like chronic injury or dermatoses (Clavel et al 1999; Cox et al 2007).In Bowen's illness, the epidermis will exhibit both parakeratosis and hyperkeratosis histopathologically. Acanthosis will also be evident, causing the rete ridges to thicken and elongate. Overly keratinocytic cells, which are frequently extremely uncommon and may even have a more peculiar appearance than SCCI, will result from these alterations. The entire thickness of the epidermis is affected by the atypia, and the keratinocytes exhibit pleomorphism, increased nuclei, and vigorous mitotic activity. Additionally, they will exhibit a loss of polarity and maturity, which will cause the epidermis to appear disorganized or "windblown." There are two different kinds of multinucleated cells that can be observed: a dyskeratotic cell that is absorbed in the cytoplasm of a keratinocyte, and a multinucleated giant cell. On rare occasions, higher epidermis cells will appear eosinophilic cytoplasm.[59]

**Pemphigus vulgaris**

A collection of uncommon autoimmune bullous disorders that affect the skin and mucous membranes are together referred to as pemphigus diseases. Two new instances per million people are reported there each year. They have a chronic progression, marked morbidity and death, and a substantial decline in quality of life. The pathogenic autoantibodies (often of the IgG class) produced against various desmosome proteins (desmogleins) cause this group of disorders to emerge. These autoantibodies against the desmosome components work together to impair intraepidermal adhesion, which causes acantholysis, vesicle development, blisters, and erosions on the skin and/or mucous membranes.

Since acantholytic keratinocytes are seen in a number of vesiculobullous disorders, a histopathological investigation of

pemphigus vulgaris aids in both diagnosing the condition and differentiating it from other subepidermal bullous lesions. Since PV blisters typically break rapidly, it is advised to select a recent blister for the biopsy (less than 24 hours from the time of emergence) that fits inside a 4 mm punch or a small fusiform excision. The degree of epidermal cleavage is indicated by histopathological analysis (suprabasal or intramalpighian). Its contents are frequently lost during histological processing or inflammatory cells, with neutrophils predominating and eosinophils finally becoming visible.

The adnexal epithelium (often the follicular epithelium) may be impacted by acantholysis, which aids in the differential diagnosis of Hailey-Hailey disease. An inflammatory infiltration is seen in the papillary dermis, with neutrophils predominating in the perivascular area.[10,60]

**Basal cell carcinoma**

The most prevalent cancer in humans is basal cell carcinoma (BCC), formerly known as basal cell epithelioma. BCC typically appears on skin that has been sunburned; it hardly ever appears on mucous membranes, the palms, or the soles. Metastases from basal cell carcinoma are uncommon since the tumor grows slowly. BCC is rarely lethal, but when therapy is insufficient or delayed, it can be extremely damaging and deform local tissues. When BCC is examined clinically, it typically manifests as flesh- or pink-colored pearly papules that have telangiectatic veins or ulceration on top of them. Sun-damaged skin is a predisposition of basal cell carcinoma (BCC), which originates from the hair follicle and/or the interfollicular epidermis.[61]

Although the literature lists over 26 different forms of BCC, the more prevalent, recognizable, and clinicopathologic varieties are: nodular, micronodular, superficial, morpheaform, infiltrative, and fibroepithelial (also called fibroepithelioma of Pinkus). It is also possible for these categories to combine. Although melanin may be present in varying levels within BCCs, the bulk of these tumors are amelanotic.

Islands or nests of basaloid cells, with cells palisading at the periphery in an ad hoc fashion in the cores of the islands, are a characteristic feature of BCCs. These tiny pleomorphic cells are made up of a sparse amount of cytoplasm and a basophilic nucleus devoid of a noticeable nucleolus. Clefting, another name for retraction artifact, is typically observed between the surrounding stroma and the tumor. Both the tumor and the surrounding stroma may have mucin deposition. Additionally, mitotic figures could exist. Perineural invasion, another name for perineural proliferation, may be a sign of an aggressive illness.[62]

Jaykaer SS et al did a study in 2013 on 85 cases of hyperpigmented histologically diagnosed cutaneous lesions to evaluate the histopathological spectrum.Of these eighty-five cases, 38 were male and 47 were female with the eldest being an eight-year-old male. More women than men were impacted. The extremities were the most often affected areas, followed by the back, face, neck, chest, abdomen, and every part of the body with several sites was involved. With 12.94% of cases, classical lichen planus was the most frequent lesion followed by variants of lichen planus (10.5%), psoriasis (5.8%), prurigo nodularis (5.8%), morphea (4.7%),lichen simplex chronicus (4.7%), lichen striatus (3.5%), verruca planus (3.5%), Becker's nevus (3.5%), pemphigus foliaceous (2.3%), linear epidermal nevus (2.3%), macular amyloidosis (2.3%), lentigo simplex (2.3%), lichen simplex et atrophicus (2.3%) and 1.1% each of lichen amyloidosis, junctional nevus, panniculitis and many others.The majority of patients had dermal abnormalities such as pigment incontinence and perivascular and interstitial mononuclear infiltration, as well as epidermal abnormalities such as hyperkeratosis, acanthosis, and basal cell degeneration, according to histological analysis.Thus, it was concluded that lichen planus was the most common diagnosis for hyperpigmented skin lesions, which are most common in females.[63]

Mruthyunjayappa et al did a prospective study from August 2012 to August 2014 to study the histopathological spectrum of hyperpigmented skin lesions in tertiary care centre in Bengaluru. Two hundred (20.4%) of the 980 skin biopsies showed hyperpigmented lesions. The second and third decades had the highest frequency of lesions. Itching was the most typical clinical manifestation. The most frequent biopsy-confirmed hyperpigmented lesions were variations of classical lichen planus, accounting for 55% of cases followed by postinflammatory hyperpigmentation (9%), lichen simplex chronicus (8%), prurigo nodularis (8%), polymorphous light eruption (8%) and morphea (6%). The majority of lesions had lichenoid/interface dermatitis on histopathology. Lichen planus pigmentosus was the only dermal hyperpigmented lesion, while the others were epidermal hyperpigmented lesions accounting for 75% of all hyperpigmented skin lesions. It was determined that the most prevalent hyperpigmented lesion observed in their investigation was classical lichen planus. Authors concluded that when diagnosing hyperpigmented skin lesions accurately, histopathological testing is helpful, especially when the lesions have overlapping or ambiguous clinical symptoms. In this study, there was a positive connection in 95% of instances and a negative correlation in 5% of the cases between the histopathological and clinical diagnoses. Discordance was basically resolved by determining the important histological characteristics that did not match the clinical diagnosis. [30]

From June 2015 to June 2016, Dr. PRD Ganesh Basina and colleagues conducted a prospective study at Rangaraya Medical College's pathology department in Kakinada. Sixty examples of cutaneous lesions with hyperpigmentation were assessed. The age range covered by this study was 5 to 75 years old. In the 5-year-old female kid, the condition was dermatofibroma, and in the 75-year-old female, it was basal cell carcinoma. With a total of 13 instances, five of which were malignant lesions, the sixth decade was the most prevalent age group. The most common type basal cell carcinoma prominent in

the sixth decade, but the fifth decade saw a higher incidence of malignant melanoma.The ratio of men to women was determined to be 1.1:1. The percentage of non-melanocytic lesions out of the 60 instances was 93.33%, whereas the percentage of melanocytic lesions was just 6.67%. According to the study's findings, inflammatory lesions made up 66.66% of all lesions, with lichen planus and its variations accounting for 20% of these cases. Melanocytic lesions made up only 20% of all neoplastic lesions; the remaining 80% were non-melanocytic lesions. While malignant melanoma accounts for 75% of cases in melanocytic neoplasms, basal carcinoma is the most prevalent entity in the nonmelanocytic neoplastic category (44%) and is followed by seborrhoeic keratosis (25%) category, with the sole benign neoplastic melanocytic lesion being lentiginosa. According to their study, nonmelanocytic inflammatory, benign, and malignant lesions made up 93.3% of the 60 pigmented cutaneous and mucosal lesions, whereas melanocytic benign and malignant lesions made up 6.6%. Consequently, it was concluded that hyperpigmented skin and mucous membrane lesions might vary from inflammatory to malignant. In this investigation, the most prevalent pigmented lesions were lichen planus and its variations.[64]

In 2018, a study was carried out by Goyal et al. to investigate the clinicopathological association of 50 cases of pigmented skin lesions. The findings showed that post inflammatory hyper pigmentation (22%) was the primary cause of pigmentary disorders of the skin. Lichen planus pigmentosus (16%), macular amyloidosis (10%), lichen amyloidosis (8%), seborrheic keratosis (8%), epidermal nevus (6%), becker's nevus (4%), dermatofibroma (4%), drug reactions (4%) and 2% of angiokeratoma, nevus of ota, and melasma were the next most common causes identified. The majority of the cases included patients between the ages of 21 and 30, with the youngest patient being 15 and the oldest being 70. With a male to female ratio of 1:1.1, a slight female preponderance was observed. Acellular eosinophilic material and increased basal cell layer pigmentation (18%), perivascular infiltrate

(72%) and pigment incontinence (62%), elongated rete ridges (30%), papillomatosis, horn cysts and psuedocysts (8%), nests of nevus cells (6%), proliferation of melanocytes (4%) and dilated blood vessels (2%), were the most common histopathological features. Authors opined thatin order to make a conclusive diagnosis in all cases of pigmented lesions that have been clinically diagnosed, histological testing should be performed.[40]

Adhikari et al conducted a retrospective study from January 2016 to December 2018 on 1040 skin biopsies to evaluate the histopathological spectrum of skin lesions in a tertiary care centre in Nepal. The age range of 31 to 40 years old comprised the majority of the patients. Non-infectious vesicobullous and vesicopustular disease account for 28.6% of all skin diseases, with non-infectious erythematous papular and squamous disease coming in second at 25.9%. Spongiotic dermatitis was the most prevalent vesicobullous condition (84.8%). The most prevalent non-infectious erythematous papular and squamous disease was Erythema dyschromicum perstans (31.8%) which was followed by Lichen planus (17.4%). Leprosy was the second most prevalent microbiological disease, behind fungal infection. The most prevalent neoplastic diseases of the skin were epidermal tumors, with basal cell carcinoma being the most common type. The most prevalent skin tumor was called a melanocytic nevus. The upper extremities were the most often involved site. Positive correlation between clinical and histological diagnosis was seen in 73.8% cases. [65]

Bohra et al in a study conducted on clinicohistopathological correlation of pigmented skin lesions in 2019 showed that 32 cases of melanocytic lesions, including benign melanocytic nevi (n = 27), malignant melanoma (n = 5), and 14 cases of nonmelanocytic lesions, including basal cell carcinoma and seborrheic keratosis (5 cases each), were among the 46 cases that were analyzed. One person each with angiokeratoma, sebaceous hyperplasia, trichoepitheloma, and venous haemangioma. The age range was 8–83 years, with a little female

predominance (52.2%) and head and neck involvement (58.7%) as the most prevalent site affected. In 26 cases (56.5%), the clinicohistopathological association was positive, whereas, in 20 cases (43.5%), it was negative.[66]

In 2020, Abhishek Singh et al conducted a study on clinicopathological spectrum of pigmented skin lesions at a tertiary care hospital's pathology department.In the research, 75 cases of pigmented skin lesions were reviewed on paraffin embedded tissue sections stained with hematoxylin and eosin between June 2017 and May 2020. The cases were analysed based on age, gender, site of occurrence, and histological type. There were 52 non-melanocytic lesions and 23 melanocytic lesions out of the 75 cases that were analysed. Benign melanocytic nevi (13 cases) were the most prevalent lesion overall, followed by Seborrheic keratosis (11 cases). Of all the benign nevi, majority (69.2%) were intradermal nevi followed by compound nevi (23.1%) and congenital melanocytic nevus (7.7%). Malignant melanoma was seen in 13.3% cases. Lesions from the first to the ninth decade were seen with majority of them being in third decade with a little female advantage. The head and neck were the most often affected areas. In 55 cases (73.3%) the clinicopathological association was positive; in 20 cases (26.6%) it was negative. The majority of pigmented skin lesions were benign, yet they frequently manifested as a problem.[67]

In 2020, Dr. Sneha Ravindran and colleagues conducted a study on hyperpigmented skin lesions, involving 100 individuals of all ages with hyperpigmented punch skin biopsy lesions. Majority of the cases were in the age group of 21-30 years (43%) with male preponderance and majority of the patients presenting with pigmented itchy macules and papules over the extremities, back and trunk. At most, 45% of cases were classical lichen planus and its variations followed by eczematoid dermatitis (12%), Polymorphous light eruption (8%), Discoid Lupus erythematosus (7%), post inflammatory hyper pigmentation (7%),lichen

simplex chronicus (6%), prurigo simplex (4%), Morphea (4%), prurigo nodularis (4%), acne (3%) and erythema dyschromium perstans (1%). Thus, it was concluded that the most prevalent hyperpigmentary skin lesions observed in the study were lichen planus along with its variations. The study's examination of the relationship between the histological and clinical diagnoses showed a positive connection in 90% of instances and a negative correlation in 10% of cases, highlighting the significance and usefulness of histology in reaching a definitive diagnosis.[68]

In terms of environmental factors that contribute to premature skin aging, Qi Yi Ambrose Wong et al's meta-analysis found that solar UV irradiation was the most significant, accounting for 80–90% of the morphological, structural, and biochemical changes that were collectively referred to as chronic cutaneous photo ageing. The most prominent extrinsic ageing component was sun exposure, which was strongly linked with several skin ageing symptoms that could be identified as being independent of ethnic origin and which showed dose-response relationships with wrinkles.[69]

Anand et al in a study conducted in 2019 to emphasize the importance of stratification of pigmented skin lesions showed that of the 70 cases that were examined, 22 (31%) had melanocytic lesions, such as malignant melanoma, which had 12 cases (17.1%), benign nevi, which had 10 cases (14.2%), and 48 (69%) had non-melanocytic lesions, such as lichen planus, which had 30 cases (42%), seborrheic keratosis, which had 10 cases (14.3%), and basal cell carcinoma, which had 8 cases (11.4%). The age range of these lesions were from 8 to 92 years old. When these lesions were examined one at a time, the distribution of these lesions began to exhibit a pattern even if it was equal in both boys and girls overall. Male: Female ratiowere different in different diseases asmales predominant in Malignant Melanoma (11:30), Seborrheic Keratosis (7:3), and Basal Cell Carcinoma (5:3), whereas females are more likely to have Lichen planus (1:2) and

Benign nevi (1:9) lesions. The extremities were more frequently affected by pigmented skin lesions than the head, neck, and trunk. 100% of occurrences of malignant melanoma occured in the extremities, 70% of cases of benign nevi occured in the head and neck, and 70% of cases of seborrheic keratosis occured in the same area. The face, head, and neck region accounted for 100% of occurrences of basal cell carcinoma, while the extremities were the most common site for lichen planus. Malignant melanomas accounted for the majority of melanocytic lesions (17.1%). The age range of 50–70 was where the majority of instances occured (66.6%). This research showed that cutaneous melanomas were increasingly emerging among the melanocytic lesions. This led to the assumption that there may be a growing trend of cutaneous melanomas in India, which was currently thought to be uncommon. The focus of this study was on the histomorphological examination of pigmented skin lesions, as this remains the primary method for identifying cutaneous melanomas.[70]

Another study, conducted in 2021 by Ahmed SA et al., was a retrospective analysis of all skin biopsy tissues that presented as pigmented lesions from January 2017 to December 2019, in the pathology department of a tertiary care centre. Five biopsies of the 58 cases of pigmented skin lesions had insufficient material, and five of them produced inconclusive results. Thirty-three of the remaining cases were malignant, and fifteen were benign. Of these cases, only eight were malignant melanoma cases and ten were nevi; the remaining 18 were all of melanocytic origin. The results of this investigation indicated that the histopathological and clinical diagnoses correlated favourably (54%). Consequently, it was concluded that proper histological diagnosis requires strong clinical correlation and biopsy.[71]

Avani et al in a study conducted in 2021 on spectrum of hyperpigmented skin lesions showed that during the allotted time of 1.5 years, 100 skin biopsies from hyperpigmented lesions were examined.

The age range of 21–40 years old accounted for 50% of the cases, with the age group of 41–60 years old accounting for 33% of cases. The oldest case was 75 years old, and the youngest was 6 years old. Females were more afflicted than males in this set of cases, with 56 % being females and 44% being males. Itching was the most prevalent clinical symptom (86%) other than hyperpigmentation. Four limbs were involved bilaterally in the majority of cases (44%), with only the upper limbs involved in 25% of cases and the entire body involved in 20% of instances. The majority of cases (38%) with histopathological evaluation were of Lichen planus and its variations, including Lichen planus hypertrophicus, Lichen planus pigmentosus, and Lichenoid-like keratosis or response. Additional cases included Prurigo nodularis, Polymorphous light eruptions (8%), Discoid lupus erythematosus (7%), Eczematous dermatitis (11%), and Post-inflammatory hyperpigmentation (6%). A single instance of Erythema dyschromicum perstans and a small number of cases of Pityriasis versicolor (3%) and Riehl's melanosis (2%) as well as Morphea and Melasma (4% each) were also noted.[72]

Goswami et al conducted a study on histopathological spectrum of skin lesions in 2022 and observed that of the 610 biopsies, 531 (87.1%) had no neoplasm, 29 (4.8%) had neoplasm, and 50 (8.1%) had no definitive results. In terms of non-neoplastic histological patterns, non-infectious vesiculobullous and vesiculopustular diseases accounted for the majority (33.3%). Seborrheic keratosis (17.7%) was the most prevalent vesiculobullous and vesiculopustular illness, followed by pemphigus (8.5%) and dermatitis (4.7%). Leprosy was the most frequent infectious condition (22.6%) followed by verruca vulgaris (4.1%). In non-infectious erythematous papular and squamous illness, lichen planus (6.2%) was the most prevalent, followed by psoriasis (5.3%). In this study, people over 60 years old were more likely to have skin lesions, and this age group was predominately male. Out of all the neoplastic lesions, basal cell carcinoma accounted for the majority with 44.8%. Squamous cell carcinoma followed with 41.4% and melanoma with 6.9%.[73]

Ranabhat et al in a study conducted in 2023 on 80 patients to study the histopathological spectrumof pigmented skin lesions showed that the patient's age was seven to eighty-eight years old, with a mean ± SD of 49.55 ± 21.73 years. The gender ratio was 1.3:1, with 34 (42.6%) males and 46 (57.4%) females. The majority of pigmented skin lesions (55%) were found on the face, followed by the trunk (18.7%), extremities (16.2%), scalp (8.8%), and genitalia (1.3%). According to a histopathological analysis, melanocytic nevi accounted for 17% of all lesions. These were followed in frequency by basal cell carcinoma (13.8%), seborrheic keratosis (6.6%), keratoacanthoma (5.3%), verrucous epidermal nevus (4%) and a small number of other instances. There was clinicopathological agreement in 44 cases (55%). This study concluded that sometimes benign lesions can be mistaken for malignant tumors. Thus, histopathological testing continues to be the gold standard for identifying these illnesses and offering appropriate direction for patients' ongoing care.[74]

# MATERIALS AND METHODS

## SOURCE OF DATA

This study was carried out in the pathology department of Dayanand Medical College and Hospital Ludhiana for a duration of 1.5 years (18 months), starting on September 15, 2022, and ending on March 14, 2024. The Department of Pathology received 21000 histopathology samples in total, 356 of which were skin biopsies. Ninety biopsies of clinically identified hyperpigmented skin lesions were among them; they were examined under a microscope, and specific stains were used as needed. These results were divided into inflammatory lesions, benign and malignant tumors based on their correlation with the clinical diagnosis.

## METHOD OF COLLECTION OF DATA

### Inclusion criteria

Skin biopsies of all the hyperpigmented lesions of the skin received in Department of Pathology, DMCH.

### Exclusion criteria

All other skin biopsies which are not hyperpigmented.

## METHODOLOGY

Skin samples of hyperpigmented skin lesions in clinically diagnosedcases were taken in the dermatology department and sent in 10% formalin to the pathology department. Biopsies were routinely treated and preserved to produce thin layers of three to four microns paraffin slices, stained with eosin and haematoxylin, and then examined under a microscope. The proforma included histological results and pertinent clinical information, such as the biopsy site and the clinical diagnosis. Wherever necessary, special stainswere used like 20% ZN, PAS and congo red.

After that, the slides were examined at 10X, 100X, and 400X magnifications. Whenever necessary, serial sectioning was carried out. It was done to interpret the histological results and correlate them with the clinical diagnosis.

**Paraffin block tissue processing**

In the automatic tissue processor, the tissue frozen in 10% formalin was run through a succession of solutions as follows:

● Acid formalin for four hours
● Formal alcohol for one hour
● 80% ethyl alcohol for one hour
● 90% ethyl alcohol for one hour
● Absolute ethyl alcohol (100%) for one hour
● Acetone (1st change) for one hour
● Acetone (2nd change) for one hour
● Xylene (1st change) for one hour
● Xylene (2nd change) for one hour
● Paraffin (1st change at 58-60°C) for one hour
● Paraffin (2nd change at 58-69°C) for one hour

Using a rotating microtome, the tissue blocks encased in paraffin were sliced to a thickness of 2-3 microns. After that, these sections were placed on slides that had been albuminized. Glass marking pencils were used in appropriately labelling these parts.

**Hematoxylin and Eosin Staining Procedure**

- To dewax the sections, slides were placed on a hot plate and heated to 60°C.
- Next, xylene was used to deparaffinize the slides.
- Next, hydration was achieved by washing them in decreasing rates of alcohol to water.
- After that, the slides were stained for 15 minutes with Harris Hematoxylin.

  After thoroughly washing under running water, the sections turned blue. After differentiating these pieces in 1% acid alcohol

(1% HCI in 70% alcohol) for 5–10 seconds, the sections were thoroughly cleaned with tap water until they turned blue once more in 5 minutes or less.

- These were once more stained for ten minutes in 1% Eosin before being cleaned for one to five minutes under running tap water.
- Sections were washed in xylene, dehydrated using increasing grades of alcohol, and mounted with DPX (distyrenedibutylphthalate xylene) to prevent air bubbles.
- After that, stained slides were examined under low (100x) and high (400x) power microscopes.

# OBSERVATIONS AND RESULTS

The present study was conducted in the Department of Pathologyat Dayanand medical college and hospital for a period of 18 months from 15 September 2022 to 14 March 2024. A total of 21000 histopathology samples were received in the department of Pathology out of which there were 356 skin biopsies. Of the 356 skin biopsies, there were 90 cases of clinically diagnosed hyperpigmented skin lesions which were included in the present study. These findings were correlated with the clinical diagnosis and categorized into inflammatory, benign and malignant lesions.

Spectrum of the hyperpigmented skin lesions withdistribution of various lesions, age and gender wise distribution, site of involvement, clinical patterns, histopathological features in different lesions and their correlation with clinical diagnosis were analysed.

**Age distribution of all lesions (n=90)**

Of the 90 cases, majority of the cases [28 cases,31.1%] of hyperpigmented skin lesions presented in third decade followed by fifth decade [16 cases,17.7%]. This study showed wide variation in age distribution with youngest being a 3 year old femaleand oldest being a 83 years old male. The mean age was 44.46 year. [Table 1]

**Table 1: Age distribution of all lesions (n=90)**

| Age group (in years) | Number of cases(n=90) | Percentage |
|---|---|---|
| < 10 | 02 | 2.2% |
| 11-20.0 | 02 | 2.2% |
| 21-30.0 | 15 | 16.7% |
| 31-40 | 28 | 31.1% |
| 41-50 | 12 | 13.3% |
| 51-60 | 16 | 17.7% |
| 61-70 | 09 | 10% |
| 71-80 | 05 | 5.5% |
| 81-90 | 01 | 1.1% |
| Total | 90 | 100.0% |

**Gender wise distribution of all lesions (n=90)**

Of the 90 cases, female preponderance was noted with 54 cases in females [60%] and 36 cases in males [40%]. The ratio of male:female is 1: 1.5 [Figure 1]

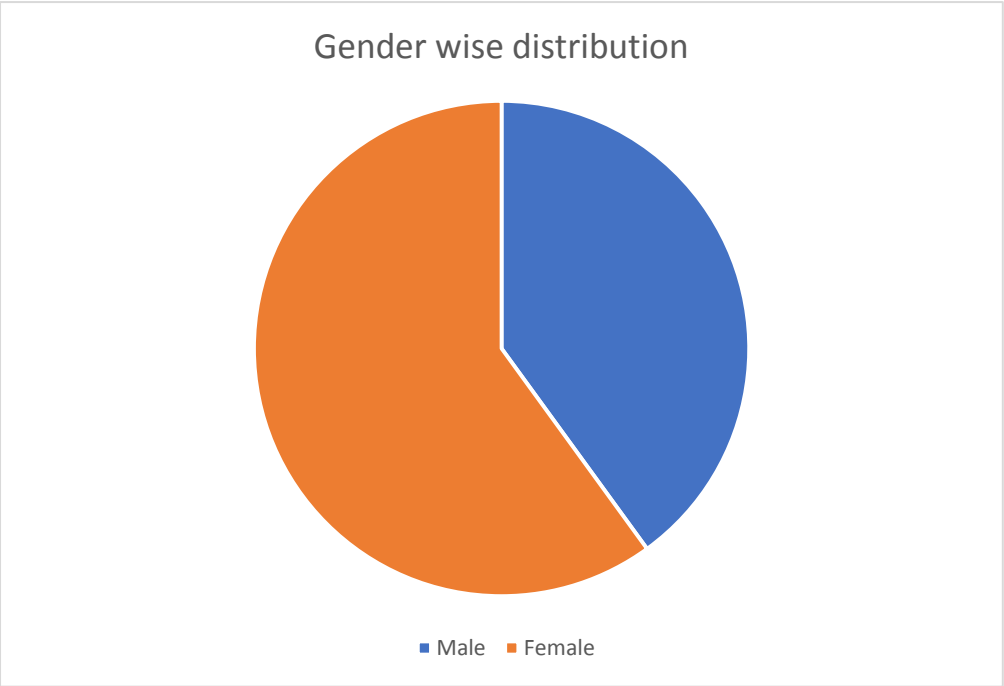


**Figure 1: Gender wise distribution of all lesions (n=90)**

**Site of involvement of all lesions (n=90)**

Of the 90 cases, most common site of hyperpigmented lesions were the limbs [n=43,47.8%], followed by lesions over the trunk and back [n=20,22.2%]. Lesions were also seen on the face and neck area [n=18,20.0%]. The least number of patients presented over the scalp [n=8,8.8%] followed by genital area (Penis) [n=1,1.1%]. [Table 2]

**Table 2: Site of involvement of all lesions (n=90)**

| Site of lesion | Number of cases(n=90) | Percentage |
|---|---|---|
| Limbs | 43 | 47.8% |
| Trunk/ Back | 20 | 22.2% |
| Face area | 18 | 20.0% |
| Scalp | 08 | 8.8% |
| Penis (Genital area) | 01 | 1.1% |

**Clinical presentation of all lesions (n=90)**

Of the 90 cases, majority[n=38,42.2%] presented as plaques followed bypapules [n=18,20.0%], patches [n=16,17.7%], macules [n=8,8.8%], nodules [n=5,5.5%], ulcers [n=3,3.3%] and reticular pattern [n=2,2.2%]were also seen.[Table 3]

**Table 3: Clinical presentation of all lesions (n=90)**

| Clinical presentation of lesions | Number of cases(n=90) | Percentage |
|---|---|---|
| Patches | 16 | 17.7% |
| Plaques | 38 | 42.2% |
| Papules | 18 | 20.0% |
| Macules | 08 | 8.8% |
| Nodules | 05 | 5.5% |
| Ulcers | 03 | 3.3% |
| Reticular pattern | 02 | 2.2% |
| Total | 90 | 100% |

**Symmetry of all lesions (n=90)**

Of the 90 lesions, most common distribution was over the bilateral limbs seen in 43 cases (47.7%). Unilateral lesions were seen in 35 cases (38.8%) followed by generalised lesions all over the body with no site predilection in 12 cases (13.3%).[Table 4]

**Table 4: Symmetry of all lesions (n=90)**

| Symmetry | Number of cases(n=90) | Percentage |
|---|---|---|
| Unilateral | 35 | 38.8% |
| Bilateral | 43 | 47.7% |
| Generalised | 12 | 13.3% |
| Total | 90 | 100.0% |

**Number of all lesions (n=90)**

Of the 90 cases of all hyperpigmented lesions, 66 cases (73.3%) presented as multiple lesions on the body while 24 cases (26.6%) presented as solitary lesion on various sites over the body. [Table 5]

**Table 5: Number of all lesions (n=90)**

| Number of Lesion | Number of cases (n=90) | Percentage |
|---|---|---|
| Multiple | 66 | 73.3% |
| Solitary | 24 | 26.6% |
| Total | 90 | 100.0% |

**Clinical diagnosis of all lesions (n=90)**

Of the 90 skin biopsies of hyperpigmented skin lesions received in Department of Pathology , majority (n=35,38.8%) of the cases came with the clinical diagnosis of lichen planus and its variants which included 22 cases of lichen planus, 9 cases of lichen planus pigmentosus and 4 cases of lichen planus hypertrophicus followed by discoid lupus erythematosus (n=13,14.4%), prurigo nodularis (n=10,11.1%), dermatitis herpetiformis (n=8,8.8%), basal cell carcinoma (n=5,5.5%),intradermal nevus (n=4,4.4%), morphea (n=3,3.3%), endogenous eczema (n=3, 3.3%), Becker's nevus (n=2, 2.2%), livedo reticularis (n=2, 2.2%), granuloma annulare (n=2, 2.2%) and malignant melanoma (n=1, 1.1%). [Table 6]

**Table 6: Clinical diagnosis of all lesions (n=90)**

| Clinical Diagnosis | Number of cases (n=90) | Percentage |
|---|---|---|
| Lichen Planus and its variants | 35 | 38.8% |
| Discoid Lupus Erythematosus | 13 | 14.4% |
| Prurigo nodularis | 10 | 11.1% |
| Dermatitis herpetiformis | 08 | 8.8% |
| Basal cell carcinoma | 05 | 5.5% |
| Intradermal nevus | 04 | 4.4% |
| Endogenous eczema | 03 | 3.3% |
| Morphea | 03 | 3.3% |
| Melanocytic nevus | 02 | 2.2% |
| Livedo Reticularis | 02 | 2.2% |
| Granuloma annulare | 02 | 2.2% |
| Becker's nevus | 02 | 2.2% |
| Malignant melanoma | 01 | 1.1% |
| Total | 90 | 100% |

All the 90 cases of hyperpigmented skin lesions were studied, of which inflammatory lesions were 68 in number (75%), benign tumors were 16 in number (17.8%) and malignant tumors were 6 in number (6.6%).

**Histological diagnosis of all hyperpigmented lesions (n=90)**

Of the total 90 cases, majority (n=29,32.2%) of the cases were diagnosed as lichen planus and its variants on histopathology followed by lichen simplex chronicus (n=10, 11.1%), discoid lupus erythematosus (n=8,8.8%), lichenoid drug eruption (n=7, 7.7%), postinflammatory hyperpigmentation (n=5,5.5%), seborrhoeic keratosis (n=4, 4.4%), verruca vulgaris (n=4,4.4%), intradermal nevus (n=4, 4.4%), basal cell carcinoma (n=4, 4.4%), Becker's nevus (n=3, 3.3%), morphea (n=3, 3.3%), prurigo nodularis (n=2,2.2%), subacute spongiotic dermatitis (n=2,2.2%), malignant melanoma (n=2,2.2%), lichen amyloidosis (n=1,1.1%), fixed drug eruption (n=1,1.1%) and verrucous epidermal nevus (n=1,1.1%). [Table 7]

**Table 7: Histological diagnosis of all lesions (n=90)**

| **Histological Diagnosis** | **Number of cases(n=90)** | **Percentage** |
|---|---|---|
| Lichen Planus and its variants | 29 | 32.2% |
| Lichen simplex chronicus | 10 | 11.1% |
| Discoid lupus erythematosus | 08 | 8.8% |
| Lichenoid drug eruption | 07 | 7.7% |
| Post inflammatory hyperpigmentation | 05 | 5.5% |
| Intradermal nevus | 04 | 4.4% |
| Seborrhoeic keratosis | 04 | 4.4% |
| Verruca vulgaris | 04 | 4.4% |
| Basal cell carcinoma | 04 | 4.4% |
| Becker's nevus | 03 | 3.3% |
| Morphea | 03 | 3.3% |
| Subacute spongiotic dermatitis | 02 | 2.2% |
| Prurigo nodularis | 02 | 2.2% |
| Malignant melanoma | 02 | 2.2% |
| Amyloidosis | 01 | 1.1% |
| Fixed drug eruption | 01 | 1.1% |
| Verrucous epidermal nevus | 01 | 1.1% |
| Total | 90 | 100% |

Hyperpigmented skin lesions were categorized into inflammatory lesions, benign and malignanttumors. Out of 90 cases of hyperpigmented lesions, 68 cases were inflammatory lesions,16 cases were benign and 6 cases were malignant tumors. Details of their age and sex distribution, site, categorization of lesions, clinical patterns and histopathological features is discussed accordingly.

**Age distribution of hyperpigmented skin lesions (n=90)**

Of the diagnosed 90 cases, 68 cases were inflammatory lesions, 16 were benign tumorsand 6 were malignanttumors. Of the 84 inflammatory lesions and benigntumors, maximum number of 28 cases [33.3%] were in the age group of 31 to 40 years. Two cases [2.3%] were among age group of 0-10 years, 2 cases [2.3%] were in age group of 11 to 20 years, 15 cases [17.8%] were 21-30 years, 14 cases [16.6%] were diagnosed in age group 51-60 years ,6 cases [7.1%] in age group 61 to 70 age group, 71 to 80 years had 4 lesions [4.7%] and only 1 case [1.1%] was seen in 81 to 90 years of age group. Among malignant tumors, maximum of 3 cases [50%] were seen in age group 61 to 70, 2 cases [33.3%] were in age group 51 to 60 years and only one case [16.6%] was seen in age group 71-80 years. [Table 8]

**Table 8: Age distribution of benign and malignant hyperpigmented skin lesions (n=90)**

| **Age** | **Inflammatory &Benign** | | **Malignant** | | **Total** |
|---|---|---|---|---|---|
| | **Number of cases** | **Percentage** | **Number of cases** | **Percentage** | |
| 10 | 02 | 2.3% | 00 | 0% | 02 |
| 11-20 | 02 | 2.3% | 00 | 0% | 02 |
| 21-30 | 15 | 17.8% | 00 | 0% | 15 |
| 31-40 | 28 | 33.3% | 00 | 0% | 28 |
| 41-50 | 12 | 14.2% | 00 | 0% | 12 |
| 51-60 | 14 | 16.6% | 02 | 33.3% | 16 |
| 61-70 | 06 | 7.1% | 03 | 50% | 09 |
| 71-80 | 04 | 4.7% | 01 | 16.6% | 05 |
| 81-90 | 01 | 1.1% | 00 | 0% | 01 |
| TOTAL | 84 | 100.0% | 06 | 100.0% | 90 |

**Gender distribution of histopathologically diagnosed hyperpigmented skin lesions (n=90)**

Of the 90 histopathologically diagnosed cases, 84 [96%] were benign and 6 [6%] were malignant. There was female preponderance in benign lesions with male: female ratio of 1:1.5. Of the 84 benign cases, 51 [60.7%] were female while 33 [39.3%] were males. There was equal distribution between males and females in case of malignant lesions [50% each] with male: female ratio of 1:1. [Table 9]

**Table 9: Gender distribution of benign and malignant hyperpigmented skin lesions (n=90)**

| **Sex** | **Inflammatory &Benign** | | **Malignant** | | **Total** |
|---|---|---|---|---|---|
| | **Number of cases** | **Percentage** | **Number of cases** | **Percentage** | |
| Female | 51 | 60.7% | 03 | 50.0% | 54 |
| Male | 33 | 39.3% | 03 | 50.0% | 36 |

**Histological spectrum of hyperpigmented inflammatory skin lesions (n=68)**

Among the 68 cases of inflammatory lesions, majority [n=29,42.6%] of the cases were of lichen planus and its variants followed by lichen simplex chronicus [n=10,14.7%], discoid lupus erythematosus [n=8,9.5%], lichenoid drug eruption [n=7,10.2%], postinflammatory hyperpigmentation [n=5,5.9%] and morphea [n=3,3.5%], prurigo nodularis [n=2,2.3%]and subacute spongiotic dermatitis[n=2,2.3%] andone case [n=1,1.1%] each of lichen amyloidosis and fixed drug eruption. [Table 10]

**Table 10: Histological spectrum of hyperpigmented inflammatory skin lesions (n=68)**

| **Final Diagnosis** | **Number of cases (n=68)** | **Percentage** |
|---|---|---|
| Lichen planus and its variants | 29 | 42.6% |
| Lichen simplex chronicus | 10 | 14.7% |
| Discoid lupus erythematosus | 08 | 11.7% |
| Lichenoid drug eruption | 07 | 10.2% |
| Post inflammatory hyperpigmentation | 05 | 7.3% |
| Morphea | 03 | 4.4% |
| Subacute spongiotic dermatitis | 02 | 2.9% |
| Prurigo nodularis | 02 | 2.9% |
| Amyloidosis | 01 | 1.4% |
| Fixed drug eruption | 01 | 1.4% |
| Total | 68 | 100.0% |

**Histological spectrum of benign tumors (n=16)**

Of the 16 benign tumors, there were 4 cases (25%) each of seborrhoeic keratosis, verruca vulgaris and intradermal nevus. There were 3 cases (18.75%) of becker's nevus and one case (6.25%) of verrucous epidermal nevus.[Table 11]

**Table 11: Histological spectrum of benign tumors (n=16)**

| Final diagnosis | Number of cases (n=16) | Percentage |
|---|---|---|
| Seborrhoeic keratosis | 04 | 25% |
| Verruca vulgaris | 04 | 25% |
| Intradermal nevus | 04 | 25% |
| Becker's nevus | 03 | 18.75% |
| Verrucous epidermal nevus | 01 | 6.25% |
| Total | 16 | 100% |

**Histological spectrum of malignant tumors (n=6)**

Of the 90 hyperpigmented lesions, 6 cases weremalignant of which 4 cases [66.6%]were of basal cell carcinoma and 2 cases [33.3%] were of malignant melanoma. [Table 12]

**Table 12: Histological spectrum of malignant tumors (n=6)**

| Final Diagnosis | Number of cases | Percentage |
|---|---|---|
| Basal cell carcinoma | 04 | 66.6% |
| Malignant melanoma | 02 | 33.3% |
| Total | 06 | 100.0% |

**INFLAMMATORY DISORDERS**

**Lichen planus and its variants**

The lesions included in lichen planus and its variants

- Classical lichen planus (n=15)
- Lichen planopilaris (n=9)
- Lichen planus hypertrophicus (n=4)
- Lichen planus pigmentosus (n=1)

Of all 29 cases of Lichen Planus and its variants, 16 [55.1%] were seen in females and 13[44.8%] in males. Age distribution ranged from 22- 74 years. The most common lesions were of classical lichen planus (n=15,41.6%) followed by lichen planopilaris (n=9,25%), lichen planus hypertrophicus (n=4, 11.1%) and lichen planus pigmentosus (n=1, 2.7%).

**Classical lichen planus (n=15)**

Histological changes seen in all 15 cases of classical lichen planus were very specific. All the cases (n=15,100%) showed band like lymphohistiocytic inflammatory infiltrate, basal cell vacuolar degeneration, saw toothing of rete ridges and hyperkeratosis followed by acanthosis, wedge shaped hypergranulosis and perivascular and periadnexal inflammatory infiltrate in 14 cases (93%), parakeratosis and pigment incontinence in 13 cases (87%), follicular plugging in 12 cases (80%).[Table 13]

**Table 13: Classical Lichen Planus (n=15)**

| **Histological changes** | **Number of cases(n=15)** | **Percentage** |
|---|---|---|
| Hyperkeratosis | 15 | 100% |
| Parakeratosis | 13 | 87% |
| Wedge shaped hypergranulosis | 14 | 93% |
| Acanthosis | 14 | 93% |
| Basal cell vacuolar degeneration | 15 | 78.0% |
| Saw toothing of rete ridges | 15 | 100% |
| Follicular plugging | 12 | 80% |
| Pigment incontinence | 13 | 87% |
| Band like lymphohistiocytic inflammatory infiltrate | 15 | 100% |
| Perivascular and periadnexalchronic infiltrate | 14 | 93% |

**Lichen Planopilaris (n=9)**

Histological changes seen in all cases (100%) of lichen planopilaris were acanthosis and inflammatory infiltrate mainly concentrated around the infundibulum of the hair follicles followed by hyperkeratosis and follicular plugging in 8 cases (89%). [Table 14]

**Table 14: Lichen Planopilaris (n=9)**

| Histological changes | Number of cases (n=9) | Percentage |
|---|---|---|
| Hyperkeratosis | 8 | 89% |
| Acanthosis | 9 | 100% |
| Follicular plugging | 8 | 89% |
| Perifollicular inflammatory infiltrate | 9 | 100% |

**Lichen planus hypertrophicus (n=4)**

All the cases (100%) showed hyperkeratosis, acanthosis, basal cell vacuolar degeneration and lymphocytic infiltrate at base of rete ridges followed by hypergranulosis in 3 cases (75%). [Table 15]

**Table 15: Lichen Planus hypertrophicus (n=4)**

| Histological changes | Number of cases (n=4) | Percentage |
|---|---|---|
| Hyperkeratosis | 4 | 100% |
| Acanthosis | 4 | 100% |
| Hypergranulosis | 3 | 75% |
| Basal cell vacuolar degeneration | 4 | 100% |
| Lymphocytic infiltrate at base of rete ridges | 4 | 100% |

**Lichen planus pigmentosus (n=1)**

One case was histologically diagnosed as lichen planus pigmentosus which showed histological features as hyperkeratosis, acanthosis, increased basal cell layer pigmentation and infiltration of papillary dermis by melanophages.

**Lichen simplex chronicus**

Of all the 10 cases, 6 (60%) were seen in males while 4 (40%) were seen in females. All of them were in the age range of 20-40 years.All thecases of lichen simplex chronicus showed histological features as hyperkeratosis, acanthosis, elongation of rete ridges and dermal fibrosis in all 10 cases (100%) followed by hypergranulosis and perivascular inflammatory infiltrate composed mainly of lymphocytes in 9 cases (90%). [Table 16]

**Table 16: Lichen simplex chronicus (n=10)**

| Histological changes | Number of cases (n=10) | Percentage |
|---|---|---|
| Hyperkeratosis | 10 | 100% |
| Hypergranulosis | 9 | 90% |
| Elongation of rete ridges | 10 | 100% |
| Acanthosis | 10 | 100% |
| Dermal fibrosis | 10 | 100% |
| Perivascular chronic inflammatory infiltrate | 9 | 90% |

**Discoid lupus erythematosus (DLE)**

Of all 8 cases of DLE, 5 [62.5%] were females and 3 [37.5%] were males. The age of these patients range from 41-70 years. These patients presented with scaly hyperpigmented plaques with aggravation on exposure to sunlight. Of all the 8 cases of DLE, all the cases showed hyperkeratotic epidermis and mild to moderate perivascular and periadnexal inflammatory infiltrate (n=8,100%). 5 cases (62.5%) showed foci of epidermal atrophy at places. Basal cell vacuolar degeneration was seen in 7 cases (87.5%). Follicular plugging and mucin deposition in the papillary dermis were seen in 6 cases (75%). Few scattered apoptotic keratinocytes within the epidermis was seen in 5 cases (62.5%).[Table 17]

**Table 17: Histological features of Discoid lupus erythematosus (n=8)**

| **Histological features** | **Number of cases (n=8)** | **Percentage** |
|---|---|---|
| Hyperkeratosis | 08 | 100.0% |
| Epidermal atrophy | 05 | 62.5% |
| Follicular plugging | 06 | 75.0% |
| Mucin deposition in papillary dermis | 06 | 75.0% |
| Basal cell vacuolar degeneration | 07 | 87.5% |
| Apoptotic keratinocytes within epidermis | 05 | 62.5% |
| Perivascular and periadnexal inflammatory infiltrate | 08 | 100% |

**Lichenoid drug eruption**

Out of the 7 cases, 4 cases [57.1%] were seen in females while 3 cases[42.8%] were seen in males. All of them were seen in the age group of 30-40 years.Histological features seen in all cases (100%) of lichenoid drug eruption were hyperkeratosis, acanthosis, basal cell vacuolar degeneration, occasional scattered apoptotic keratinocytes in the epidermis and infiltration of the dermis and epidermis by inflammatory infiltrate comprising of lymphocytes, plasma cells and occasional eosinophils. Focal parakeratosis was seen in 6 cases (86%). [Table 18]

**Table 18: Lichenoid drug eruption (n=7)**

| **Histological changes** | **Number of cases (n=7)** | **Percentage** |
|---|---|---|
| Hyperkeratosis | 7 | 100% |
| Acanthosis | 7 | 100% |
| Parakeratosis | 6 | 86% |
| Basal cell vacuolar degeneration | 7 | 100% |
| Apoptotic keratinocytes in epidermis | 7 | 100% |
| Dermal infiltrate comprising of eosinophils and lymphocytes | 7 | 100% |

**Postinflammatory hyperpigmentation [PIH] (n=5)**

Of all 5 cases of PIH, 3 [60%] were seen in females and 2 [40%] in males. The age span was from 41 to 60 years. Five cases were histologically diagnosed as postinflammatory hyperpigmentation with four cases clinically diagnosed as lichen planus pigmentosus and one case as lichen planus. On histopathology, hyperkeratotic epidermis was seen in 3 cases while other 2 cases showed unremarkable epidermis. Basal cell vacuolar degeneration with increased

pigmentation was seen in all the cases [100%]. Dermis showed pigment incontinence in all the cases [100%]. 4 cases showed mild perivascular lymphocytic inflammatory infiltrate [ 80%]. However, no band like inflammatory infiltrate or any other specific features of any lesion was seen.

**Morphea (n=3)**

Of all 3 cases, 2 [67%] were females and one [33%] was male. Age range was from 30-60 years.Of 3 cases of morphea, epidermis was insignificant with mild acanthosis seen in 1 case [33.3%]. Dermis showed thickened collagen bundles in 100% of the cases with extension into the subcutaneous tissue. Mild infiltration of the papillary dermis by lymphoplasmacytic inflammatory infiltrate was seen in 1 case [33.3%]. Increased pigmentation in the basal layer of the epidermis was seen in 2 cases [66.6%].

**Prurigo nodularis (n=2)**

Of both the cases, one was a 40 year old female and another one was 50 year old female. Both presented with intense itching. Age ranges from 31-50 years.Both the cases showed marked hyperkeratosis and acanthosis of epidermis [100%]. Dermis showed mild infiltration by lymphocytes and plasma cells. The characteristic feature seen in both these cases was hypertrophy and proliferation of dermal nerves [100%].

**Subacute spongiotic dermatitis (n=2)**

Both the patients were males [100%] with one being a 34 year old and another being a 38 year old.Both the cases showed acanthotic and parakeratotic epidermis with mild to moderate spongiosis. Upper layers of the epidermis showed scattered lymphocytes, plasma cells and neutrophils. Dermis showed sparse perivascular lymphocytic inflammatory infiltrate.

**Amyloidosis (n=1)**

A 40 year old female patient presented to the dermatologist with a gradual increasing brownish papule since last one month associated with mild itching. Skin biopsy showed acanthotic epidermis with papillary dermis showing deposition of eosinophilic material. Congo red

stain with apple green birefringence under polarized microscopy confirmed the deposited material to be amyloid.

**Fixed drug eruption (n=1)**

A 48 year old female patient presented to the dermatologist with blackish discolouration of the right arm since last 5-6 months associated with mild pain. Skin biopsy showed hyperkeratotic and parakeratotic epidermis with occasional scattered dyskeratotic keratinocytes in the epidermis. Prominent basal cell vacuolar degeneration noted. Dermis shows inflammatory infiltrate comprising of lymphocytes, histiocytes and occasional eosinophils around the blood vessels.

**BENIGN TUMORS**

**Melanocytic lesions**

Of all the 8 cases, 6 [75%] were seen in females and 2 [25%] were in males. The dominant age group was 21-40 years.

Of the 8 benign melanocytic hyperpigmented tumors comprising of intradermal nevus, verrucous epidermal nevus and becker's nevus, all the 8 cases [100%] showed hyperkeratosis,elongated rete ridges with perivascular inflammation. Nevus cell were seen in dermis in 4 cases [50%]. Four cases [50%] showed hypergranulosis. Orthokeratosis was seen in 2 cases [25%] while papillomatosis was seen in one case [12.5%]. Three cases [37.5%] showed acanthosis and increasedbasal cell layer pigmentation. Vacuolated cells were seen in upper layers of epidermis in one case [12.5%]. No junctional activity/ significant mitosis/ nuclear atypia was seen in any of the cases examined. [Table 19]

**Table 19: Histological features of benign melanocytic lesions (n=8)**

| Histological features | Number of cases(n=8) | Percentage |
|---|---|---|
| Unremarkable epidermis | 01 | 12.5% |
| Thinned out epidermis | 02 | 25.0% |
| Hyperkeratosis | 08 | 100% |
| Parakeratosis | 03 | 37.5% |
| Orthokeratosis | 02 | 25.0% |
| Acanthosis | 03 | 37.5% |
| Papillomatosis | 01 | 12.5% |
| Hypergranulosis | 04 | 50.0% |
| Elongated rete ridges | 08 | 100% |
| Nevus cells in dermis | 04 | 50.0% |
| Perivascular infiltrate | 08 | 100% |
| Increased basal cell pigmentation | 03 | 37.5% |
| Vacuolated cells in epidermis | 01 | 12.5% |

**Seborrhoeic keratosis (n=4)**

Of all the 4 cases of seborrhoeic keratosis, 3 [75%] were seen in females and one case [25%] was seen in male. The age range was from 55-75 years. These patients presented with gradually increasing blackish plaque over a period of 6 months to 1 year. All the 4 cases had past history of aggravation of symptoms of itching after exposure to sunlight. 100% of the cases showed hyperkeratosis, acanthosis and papillomatosis. Horn cysts were seen in 3 cases [75%]. The epidermis showed proliferation of basaloid cells in 100% of the cases along with squamous cell proliferation in most of the cases. Dermis showed moderate infiltration by lymphocytes and plasma cells in 100% of the cases.

**Verruca vulgaris (n=4)**

Of all 4 cases of verruca vulgaris, 3 [75%] were females and one [25%] was male.The age distribution was between 31-50 years. These patients presented as hyperkeratotic papules over the skin which developed slowly over period of 2-3 years. These patients were later found to be HPV positive. Of all the 4 cases, two had lesions over feet and other two had lesions on hands. On histopathological examination, all the 4 cases[100%]showed acanthosis, hyperkeratosis, papillomatosis of the epidermis, elongation of rete ridges and koilocytes in upper layers of epidermis.

**MALIGNANT TUMORS**

**Basal cell carcinoma (BCC)**

Of all 4 cases of BCC, 2 were females and 2 were males showing an equal distribution of 50%. The age range was from 51-70 years.

Of the 4 cases of malignantlesions of basal cell carcinoma, all the cases showed orthokeratosis, nuclear pleomorphism, atypical basaloid cells, increased basal cell pigmentation and perivascular inflammation [100%]. Out of total 4 cases, hyperkeratosis and parakeratosis was seen in 3 cases [75%]. All the four cases were diagnosed as nodular variant of basal cell carcinoma as the lesions were relatively circumscribed masses clinically and were seen in the head and neck area. Pigment incontinence was also seen which is also a feature of nodular variant. [Table 20]

**Table 20: Histopathological features of Basal cell carcinoma (n=4)**

| Histopathological features | Number of cases (n=4) | Percentage |
|---|---|---|
| Hyperkeratosis | 03 | 75% |
| Orthokeratosis | 04 | 100% |
| Parakeratosis | 03 | 75% |
| Nuclear pleomorphism | 04 | 100% |
| Atypical basaloid cells | 04 | 100% |
| Increased basal layer pigmentation | 04 | 100% |
| Perivascular inflammation | 04 | 100% |

**Malignant melanoma (n=2)**

Both the patients were males [100%], one was 57 years old while another was 71 years old. The lesions were present over foot and leg in both the cases. Skin biopsy showed foci of epidermal ulceration in one case with dense inflammatory granulation, acute and chronic. The adjacent epidermis in both the cases showed hyperkeratosis, parakeratosis and acanthosis. Dermis show a tumor with tumor cells arranged in nests. Melanin pigment is seen around these nests and within cytoplasm of few cells. Few of the cells show prominent nucleoli as well. One case showed tumor cells extending upto the reticular dermis sparing the subcutaneous tissue giving Clark's classification for melanoma as level IV. Another case showed tumor cells only in the papillary dermis giving Clark's classification for melanoma as level II.

**CORRELATION BETWEEN CLINICAL AND HISTOPATHOLOGICAL EXAMINATION OF HYPERPIGMENTED SKIN LESIONS (n=90)**

Of the 90 hyperpigmented cases, 63 cases (70%) showed histopathological correlation with the clinical diagnosis while 27 cases (30%) were diagnosed only on histopathological examination. [Table 21]

**Table 21: Correlation between clinical and histopathological examination of hyperpigmented skin lesions (n=90)**

| Correlation between clinical and histopathology | Clinical and histopathological correlation | Diagnosed only on histopathology |
|---|---|---|
| Total number of cases(n=90) | 65 (72.2%) | 25 (27.7%) |

The study examined the correlation between histological and clinical diagnoses, and discrepancies were resolved by determining the key histopathological characteristics that did not match the clinical diagnosis. Ten cases were diagnosed as lichen simplex chronicus

which showed hyperkeratosis, acanthosis, dermal fibrosis and mild perivascular inflammatory infiltrate on histological examination came with the clinical diagnosis of verruca vulgaris (1 case), mycosis fungoides (1 case), lichenoid rash (1 case),lichen amyloidosis (1 case),Bowenoid papulosis (1 case), fixed drug eruption (1 case), psoariasis (1 case) and sarcoidosis (1 case).Endogenous eczema was the clinical diagnosis in 2 cases which were histologically also diagnosed as lichen simplex chroniocus showing concordance in 2 out of 10 cases. Four cases were histologically diagnosed as discoid lupus erythematosus with histological features seen as basal cell vacuolar degeneration with mucin deposition in the papillary dermis came with the clinical diagnosis as three cases of lichen planus pigmentosus and one case of sarcoidosis. Three cases were histologically diagnosed as lichenoid drug reaction with one case clinically diagnosed as psoriasis, another as basal cell carcinoma and one as drug reaction with eosinophilia and systemic symptoms showed band like inflammatory infiltrate with basal cell vacuolar degeneration and occasional scattered apoptotic keratinocytes in the epidermis. Five cases were histologically diagnosed as postinflammatory hyperpigmentation with four cases clinically diagnosed as lichen planus pigmentosus and one case as lichen planus. Histological examination showed increased basal layer pigmentation and pigment incontinence. Two cases were histologically diagnosed as subacute spongiotic dermatitis with clinical diagnosis of one as prurigo nodularis and another as plane warts. On histological examination, mild to moderate spongiosis in the epidermis with sparse perivascular inflammatory infiltrate was seen. One case with clinical diagnosis as lichen planus pigmentosus was histologically diagnosed as lichen planopilaris which showed follicular plugging and perifollicular inflammatory infiltrate. One case with clinical diagnosis as livedo reticularis was histologically diagnosed as fixed drug eruption due to absence of endothelial proliferation with deposition of fibrin in the dermal vessels and presence of basal cell vacuolar proliferation, dyskeratotic keratinocytes and vascular wall thickening with infiltration by lymphocytes, plasma cells and eosinophils.

# DISCUSSION

Pigmentation of the human skin and melanin synthesis are immenselyfluctuating, and are influenced by genetics, ultraviolet exposureand a handful of drugs. Patients' physical appearance, psychology and social functioning are all affected by a great number of skin conditions which lead to pigmentary abnormalities. There are two major types of skin pigmentation. Hyperpigmentation is a condition in which the pigment is in excess and hypopigmentation is the condition where pigment is reduced. Albinism, vitiligo, melasma, Addison's diseaseand post-inflammatory hyperpigmentation, which can be spawned by eczema, acne vulgaris, and drug interactions are considered to be the most common skin pigmentation disorders in clinical practice. Hyperpigmentation can be seen in congenital or acquired pattern which can be secondary to cutaneous or systemic disorders. Emotional distress also leads to increased pigmentation of the skin. Medicationsinhibiting tyrosinase, anti-inflammatory medications and antioxidants prevent the production of melanin, are all given as possible treatments for pigmentation problems.[75]

The skin is shielded from sunburn by the dark brown pigment melanin, which absorbs ultraviolet radiation from the sun. Dark hued individuals show higher levels of melanin, while light hued individualspertain to lower levels of melanin. The capacity of the melanin in prevention of skin cancer is one of its perks. Few studies in the literature have shown that individuals with higher levels of melanin have a less likely chance of developing skin cancer as compared to people with lower levels. Melanin also helps in regulating body temperature by absorbing the solar heatand keeping the body cool.[76]

The present work was a prospective study carried out on 90 patients who underwent clinical and histopathological examination for hyperpigmented skin lesions. In this study, the histological evaluation of hyperpigmented lesions was done and were correlated with clinical profile.

In the present study, the most common risk factor associated with the development of hyperpigmented skin lesions was sun exposure (68%) which is comparable with other studies.

Of the 90 hyperpigmented skin lesions included in thisstudy, 84 cases (94%) were benign/ inflammatory conditions while 6 cases (6%) were malignant. These malignant lesions developed in old age patients which can be attributed to the increased exposure of exposed skin to the aggravating factors like sunlight, cosmetics and other environmental factors.

The present study showed dominance of benign lesions over the malignant ones. Similar results were also seen by Abhishek Singh et al[67]and Ganesh Basina et al [64] showing 77% and 86% benign lesions in their respective studies.

***Gender wise distribution of all lesions***

Of all the 90 patients in the present prospective study, 54 were females and 36 were males with male to female ratio of 1:1.5. There were significantly greater number of females in the study. Benign lesions also showed female preponderance with male:female ratio of 1.0:1.5,whereas, malignant lesions showed an equal distribution among males and females giving female: male ratio of 1:1.

Various studies including the present study show a female preponderance in the non-neoplastic lesions with male: female ratio being 1:1.5 which is almost similar to study conducted by Krishma Goyal et al[40], Shushan SJ et al[63] and Mruthyunjayappa et al[30] who reported male: female ratio as 1:1, 1:1.2and 1:1.2 respectively in their studies. The explanation for the same could be attributed to the fact that females are more particular about skin pigmentation than males.

***Age distribution of all lesions***

The age range of the patients included in the present study was from 3 to 83 years with a peak in fourth and sixth decade of life. The mean age of the patients with benign skin lesions was 43.88 years while in the patients with malignant skin lesions was 66.33 years. These results are comparable with the observations made by other authors. The high incidence of hyperpigmented skin lesions was in the age group of 31-

40 years which can be explained by longduration of exposure of the skin to sun rays. Most patients exhibit a long history of exposure to ultraviolet radiation and sunlight that stimulates melanin production.

The present study showed 31.1% of the benign/ non neoplastic lesions in fourth decade followed by sixth decade (17.7%). Gundalli S et al[11] (60.3%), Al Saif et al[4](48%), Adhikari RC et al[65] (60%) also showed majority of the cases in the fourth decade.

***Site of involvement of all lesions***

In the present study, limbs and extremities were the most frequent site of presentation of non-neoplastic skin lesions accounting for 47.7% of the cases. Studies done by Krishma Goyal et al[40](30%) and Mruthunjayappa et al[30] (49%) also showed most of the lesions over the extremities.

***Clinical presentation of all lesions***

In the present study, most of the patients presented with hyperpigmented itchy plaques (42.2%), papules (20%) and patches (17.7%). Similar results were also seen byMruthjunyappa et al[30]in a study in which 94% hyperpigmented lesions presented as plaques followed by 64% papules and 27.5% patches.

***Histological spectrum of hyperpigmented lesions***

Of the total (histologically and clinically diagnosed) 90 cases of hyperpigmented skin lesions, 68 (75%) cases were inflammatory lesions, 16 (17.8%) cases were benign tumors and 6 (6.6%) cases were malignant tumors.

Among the 68 cases of inflammatory lesions, majority [n=46,67.4%] of the cases were of lichen planus and its variants followed by discoid lupus erythematosus [n=8,9.5%] and post inflammatory hyperpigmentation [n=5,5.9%]. Three cases [n=3,3.5%] of morphea were seen. Two cases [n=2,2.3%] each of prurigo nodularis and subacute spongiotic dermatitis were seen. One case [n=1,1.1%] each of lichen amyloidosis and fixed drug eruptionwere seen. This similar predominance of lichen planus and its variants was also seen by Shushan J et al and Mruthyunjayappa et al.

**Table 22: Comparison of final diagnosis of inflammatory lesions**

| Final Diagnosis of inflammatory lesions | Present study (n=90) | Shushan J et al[63] (n=85) | Mruthyunjayappa et al[30] (n=200) |
|---|---|---|---|
| Lichen planus and its variants | 52.9% | 23.5% | 57.5% |
| Lichen simplex chronicus | 14.7% | 4.7% | 8% |
| Discoid lupus erythematosus | 11.7% | 1.17% | 1.5% |
| Post inflammatory hyperpigmentation | 7.3% | 1.17% | 9% |
| Morphea | 4.4% | 4.7 % | 6% |
| Subacute spongiotic dermatitis | 2.9% | 4.7% | - |
| Prurigo nodularis | 2.9% | 5.88% | 8% |
| Amyloidosis | 1.4% | - | - |
| Fixed drug eruption | 1.4% | 1.17% | - |

Of the 16 benign tumors, there were 4 cases (25%) each of seborrhoeic keratosis, verruca vulgaris and intradermal nevus. There were 3 cases (18.75%) of becker's nevus and one case (6.25%) of verrucous epidermal nevus. These findings were seen in comparison with other studies in the literature.

**Table 23: Comparison of final diagnosis of benign tumors**

| Final diagnosis of benign tumors | Present study (n=90) | Abhishek Singh et al[67] (n=75) | GundalliS et al[11] (n=13) | Goel et al[77](n=232) |
|---|---|---|---|---|
| Seborrhoeic keratosis | 25% | 14.7% | 15.3% | 19.5% |
| Verruca vulgaris | 25% | - | 30.7% | 20.1% |
| Intradermal nevus | 25% | 17.3% | 84.6% | 73.8% |
| Becker's nevus | 18.75% | - | - | - |
| Verrucous epidermal nevus | 6.25% | - | - | - |

Six cases weremalignant of which 4 cases [66.6%]were of basal cell carcinoma and 2 cases [33.3%] were of malignant melanoma. These findings were analysed in comparison to other studies. Abhishek Singh et al showed a predominance of malignant melanoma (13.3%) over basal cell carcinoma (9.4%) in his study done on 75 cases. However, Goel et al showed a dominance of basal cell carcinoma in 33.3% of the total 232 cases followed by 11.9% of malignant melanoma cases.

***LICHEN PLANUS AND ITS VARIANTS***

Among all the hyperpigmented lesions including inflammatory, benign and malignant, lichen planus and its variants were most common constituting 51.1% of the total lesions.

Lichen planus is a skin condition involving mucous membranes, hair follicles and nails. These lesions clinically present as violaceous patches, papules and plaques on the flexor surface of forearms and legs. Most of the patients present with associated itching. It is categorized into various subtypes seen as lichen planopilaris, lichen planus hypertrophicus, lichen planus pigmentosus, atrophic, eruptive, annular, lichen planus actinicus and ulcerative. Drug related lichenoid reactions are also seen.[10]

In the present study, benign/non neoplastic lesions constituted 94% of the total cases. Histopathologically, most of the inflammatory lesions showed lichenoid tissue reaction /interface dermatitis.

In the present study, it was observed that classical lichen planus and its variants were the most common inflammatory hyperpigmented skin lesions constituting 32.2% of the total 90 cases in which classical lichen planus was the predominant lesion (n=15,16.6%), followed by lichen planopilaris (n=9,10%), lichen planus hypertrophicus (n=4, 4.4%) and lichen planus pigmentosus (n=1, 1.1%).It is comparable to the study done by Sneha Ravindran et al[68] which showed lichenoid lesions as the most commonlesion (45%) amongst which classical lichen planus constituted 26.7%. A similar dominance of classical lichen planus was observed by Ganesh Basina et al[64] and Shushan SJ et al[63] accounting for 20% and 12.94% respectively.

Typical histopathological features include hyperkeratosis, wedge shaped hypergranulosis, acanthosis, basal cell vacuolar degeneration and band like dermal lymphohistiocytic infiltrate. Various subtypes of lichen planus show different features along with interface dermatitis. These findings are corroborated by study conducted by Gillian Weston et al[78] in 2015.

Lichen planopilaris show dense band like perifollicular inflammatory infiltrate at the level of infundibulum and isthmus. Basal cell vacuolar degeneration at the level of infundibulum are also seen in addition to interface dermatitis.[10]

Lichen planus hypertrophicus is more commonly seen on the shins and show extensive skin thickening with increased acanthosis and hyperkeratosis.[10]

**Table 24: Comparison of histological features of lichen planus and its variants**

| Histological features | Present study (n=29) | Shushan et al[63] (n=85) | Mruthyunjayappa et al[30] (n=115) | Rohit Rampal et al[80] (n=60) | Asmita Parihar et al[79] (n=145) |
|---|---|---|---|---|---|
| Hyperkeratosis | 97.8% | 63.6% | 53.0% | 63.3% | 100% |
| Acanthosis | 97.8% | 49.5% | 27.8% | 85.0% | 94% |
| Basal cell vacuolar degeneration | 56.5% | 30.6% | 95.6% | 81.7% | 85% |
| Wedge shaped hypergranulosis | 56.5% | 15.3% | 16.5% | 61.7% | 96.5% |
| Saw toothing of rete ridges | 54.3% | - | 12.1% | 85.0% | 76% |
| Band like lymphohistiocytic inflammatory infiltrate | 56.5% | 13.0% | 55.6% | 71.7% | 94% |
| Pigment incontinence | 30.4% | 30.6% | 80.8% | 41.7% | 99% |
| Perivascular and periadnexal inflammatory infiltrate | 65.2% | 50.5% | 89.5% | 28.3% | - |
| Dermal fibrosis | 21.7% | - | 13.0% | - | 17.6% |
| Perifollicular inflammatory infiltrate | 19.5% | - | - | - | 19.3% |

All the cases of classical lichen planus and its variantsshowed common histological features like hyperkeratosis, acanthosis, basal cell vacuolar degeneration with band like lymphohistiocytic inflammatory infiltrate which was comparable to the study done by Mruthyunjayappa et al[30] in 2017 which showed similar results in a study done on 200 patients with 115 cases of lichen planus and its variants. Shushan et al[63] also showed various histopathological changes of the epidermis and dermis of the various hyperpigmented skin lesions.

Another study done by Asmita Parihar et al[79] in 2014 on lichen planus and its variants showed orthokeratosis (100%), acanthosis (94%), saw toothing of rete ridges (76%), wedge shaped hypergranulosis (96.5%) andband like dermal infiltrate (94%). The infiltrate was composed almost entirely of lymphocytes, few histiocytes and plasma cells. Civatte bodies or necrotic keratinocytes were seen in 82% of cases in the lower epidermis. Pigment incontinence was seen in 99% cases. Max Joseph spaces were apparent in 29.5% cases. These findings are comparable to the present study which showed hyperkeratosis, saw toothing of rete ridges and band like lymphohistiocytic infiltration in 100% cases followed by acanthosis, wedge shaped hypergranulosis and perivascular and periadnexal inflammatory infiltrate in 93% cases, parakeratosis and pigment incontinence in 87% cases, follicular plugging in 80% cases and civatte bodies and max joseph space were seen in 6.7% cases.

Asmita Parihar et al demonstrated thatall the cases of lichen planus pigmentosus (100%) showed epidermal thinning and pigment incontinence and 85% cases showed basal layer vacuolar degeneration. Similar features were seen in the present study with histological features as hyperkeratosis, acanthosis, increased basal cell layer pigmentation and infiltration of papillary dermis by melanophages in 100% of the cases. All the cases (100%) of lichen planopilaris histologically showed orthokeratosis, wedge shaped hypergranulosis, perifollicular infiltrate and basal layer vacuolation, 59% had civatte bodies, 23.5% showed follicular plugging and 17.6% showed perifollicular fibrosis. Follicular plugging was seen in 23.5%

patients. The present study showed all cases (100%) of lichen planopilaris exhibiting acanthosis and inflammatory infiltrate mainly concentrated around the infundibulum of the hair follicles followed by hyperkeratosis and follicular plugging in 89% cases.[79]

Rohit Rampal et al[80] in a study done in 2018 showed maximum cases of classical lichen planus with hyperkeratosis (63.3%) and band-like lympho-histiocytic infiltrate at the dermal-epidermal junction (71.7%) with basal cell vacuolar degeneration of the epidermis (81.7%). Civatte bodies (45%) and Max Joseph spaces (16.7%) were also seen in few cases. Acanthosis and saw-toothing of rete ridges (85%) were also seen.

Bangaru H et al[81] in a study conducted on 50 patients of lichen planus in 2016 showed hyperkeratosis (86%), hypergranulosis (86%), acanthosis (78%), saw toothing of rete ridges (59%) and liquefaction degeneration of basal cells (73%) along withcivatte bodies in 10% of cases, band like infiltrate (89%) and melanin incontinence (77.7%).

***LICHEN SIMPLEX CHRONICUS***

Of all the 10 cases, lichen simplex chronicus accounted for 11.1% of the total cases. Six (60%) were seen in males while 4 (40%) were seen in females. All the 10 cases of lichen simplex chronicus showed histological features as hyperkeratosis, acanthosis, elongation of rete ridges and dermal fibrosis in all 10 cases (100%) followed by hypergranulosis and perivascular inflammatory infiltrate composed mainly of lymphocytes in 9 cases (90%).

It is a prototype of chronic dermatitis and is seen as hyperpigmented lesion due to excessive scratching. Histologically, these lesions show saw toothing of rete ridges with vertical orientation of collagen bundles with perivascular inflammatory infiltration comprising of lymphocytes and plasma cells.[10]

Shushan J et al[63] had 4.7% cases of lichen simplex chronicus. Mruthyujayappa et al[30] showed 8% cases of lichen simplex chronicus with histological features seen as hyperkeratosis (67%), parakeratosis (40%), hypergranulosis (27%), acanthosis (87%) with elongation of rete

ridges (100%). Dermis showed vertically oriented collagen bundles (100%) with perivascular inflammatory infiltrate (93%).

***DISCOID LUPUS ERYTHEMATOSUS (DLE)***

Discoid lupus erythematosus shows multiple well circumscribed erythematous to hyperpigmented plaques with occasional ones exhibiting scarring. Majority of the patients present with lesions in the head and neck area. However, in cases with disseminated DLE, lesions are also present on trunk and upper limbs.[10]

Of all the 8 cases of DLE, 5 [62.5%] were females and 3 [37.5%] were males. The age of these patients ranged from 41-70 years. These patients presented with scaly hyperpigmented plaques with aggravation on exposure to sunlight. All the 8 cases of DLE showed hyperkeratotic epidermis and mild to moderate perivascular and periadnexal inflammatory infiltrate (n=8,100%). Five cases (62.5%) showed foci of epidermal atrophy at places. Basal cell vacuolar degeneration was seen in 7 cases (87.5%). Follicular plugging and mucin deposition in the papillary dermis were seen in 6 cases (75%). Few scattered apoptotic keratinocytes within the epidermis was seen in 5 cases (62.5%).

Histopathological diagnosis of DLE is based on combination of various histological features seen as hyperkeratosis, follicular plugging, lymphoplasmacytic infiltrate at dermo-epidermal junction and this infiltrate sometimes extend into the underlyingsubcutaneous tissue. Histopathological spectrum of subsets of lupus erythematosus exhibitedbasal cell vacuolar degeneration in most cases.[82] Comparing hypertrophic lupus erythematosus and squamous cell carcinoma show characteristic interstitial mucin deposition in the stroma which helps to differentiate the former from the later.[83]This histological feature was also seen in 75% cases of discoid lupus erythematosus included in the present study.

McDaniel B et al[84] in their updated study in 2023 showed DLE was more common in females of fourth and fifth decade which is in concordance to the present study.

Chanprapaph K et al[85]in their study in 2021 showed histological features of cutaneous lupus erythematosus as superficial perivascular inflammatory infiltration (42.9%, p = 0.004) however,superficial cutaneous lupus erythematosus (SCLE) and Chronic cutaneous lupus erythematosus (CCLE) with or without Systemic lupus erythematosus exhibited superficial or deep perivascular inflammatory infiltrate composed of lymphocytes (p \0.001) and periadnexal infiltration (p = 0.004). SCLE manifested the highest percentage of focal hypergranulosis (25%, p = 0.036). Specimens from CCLE patients had eminent hyperkeratosis with or without follicular plugging (72.4%, p = 0.004) (Fig. 3b). Lobular panniculitis and hyalinized sclerosis were observed solely in CCLE lesions and lupus panniculitis sections (p = 0.041). These results are comparable to the present study which showed prominent hyperkeratosis and perivascular and periadnexal infiltration in 100% of the cases. Basal cell vacuolar degeneration was seen in 87.5% in present study, however, Chanprapaph et al showed it in 68.2% of the cases.

Dewi PF et al[86] in a case study on DLE in 2021 explained microscopic examination of H&Estained section of skin biopsy (100%) revealing orthokeratosis with epidermal atrophy at places, follicular plugging and hydropic degeneration of basal cells in the epidermis. At the same time, there was visible edema and mucin in the dermo-epidermal area in the dermis, pigment incontinence in the upper dermis, and periadnexal inflammatory cell infiltrate. These findings are comparable to the present study which showed similar findings.

Hejazi EZ et al[87]and Dewi et al in their study in 2016 and 2021respectively showed similar microscopic features with the patients showing aggravation of symptoms on exposure to sunlight, and never using any sun protection. This played an essential role in establishing the role of environmental factors, especially sun exposure in the pathogenesis of DLE. The aggravation of lesions on exposure to sunlight was also seen in all cases of DLE in the present study. Haber JS et al[88] in their study conducted in 2017 also established the positive correlation between sun exposure and lupus erythematosus.

***LICHENOID DRUG ERUPTION***

Lichenoid drug eruptions are reactions that resemble lichen planus in a clinical setting, and they are experienced in response to a diverse spectrum of medications consumed. Histopathologically, focal parakeratosis and minor basal vacuolar alterations with a few eosinophils and plasma cells are distinguishing characteristics of lichenoid eruptions. Compared to lichen planus, there is a greater degree of melanin incontinence and a less dense and band-like dermal infiltrate.[30]

Out of the 7 cases, 4 cases [57.1%] were seen in females while 3 cases [42.8%] were seen in males in the present study. All of them were seen in the age group of 30-40 years. Histological features seen in all cases (100%) of lichenoid drug eruption were hyperkeratosis, acanthosis, basal cell vacuolar degeneration, occasional scattered apoptotic keratinocytes in the epidermis and infiltration of the dermis and epidermis by inflammatory infiltrate comprising of lymphocytes, plasma cells and occasional eosinophils. Focal parakeratosis was seen in 6 cases (86%).

Mruthyunjayappa et al[30] in their study showed 6 cases (3%) of lichenoid drug eruption of which 100% cases showed basal cell vacuolar degeneration and perivascular infiltrate comprising of eosinophils and plasma cells. Hyperkeratosis was seen in 67% of the cases. Half of the cases showed acanthosis and pigment incontinence.

***POSTINFLAMMATORY PIGMENTATION***

Post inflammatory hyperpigmentation often develops following cutaneous inflammation.[10] We had 5 cases of PIH, 3 [60%] were seen in females and 2 [40%] in males. The age range was from 41-60 years.Five cases were histologically diagnosed as postinflammatory hyperpigmentation with four cases clinically diagnosed as lichen planus pigmentosus and one case as lichen planus. On histopathology, hyperkeratotic epidermis was seen in 3 cases while other 2 cases showed unremarkable epidermis. Basal cell vacuolar degeneration with increased pigmentation was seen in all the cases [100%]. Dermis

showed pigment incontinence in all the cases [100%]. Four cases showed mild perivascular lymphocytic inflammatory infiltrate [80%]. However, no band like inflammatory infiltrate or any other specific features of any lesion was seen.

Maghfour et al[89] in a study conducted in 2022 proposed that an increase in melanin synthesis and distribution of melanin within the basal layer of the epidermis and dermis is the hallmark feature of this condition, however, the exact pathogenesis underlying PIH are not completely understood.

Lawrence E et al [90] in their updated study in 2022 on post inflammatory hyperpigmentation showed that it was a chronic condition which can be seen in any age with no sex predilection. Histopathological examination showed increased melanin in keratinocytes with pigment incontinence in dermis. These similar findings were seen in the present study.

Mruthyanjayappa et al[30] demonstrated pigment incontinence with dermal fibrosis in 83% of cases of postinflammatory hyperpigmentation. However, dermal fibrosis was not seen in any of the included cases of post inflammatory hyperpigmentation in the present study.

The cases included in the present study came with the clinical diagnosis of lichen planus and lichen planus pigmentosus in majority of the cases but the histological features were not consistent with the clinical diagnosis. Instead, only increased basal cell layer pigmentation and pigment incontinence was seen. Thus, confirming the theory of an underlying inflammatory condition.

***PRURIGO NODULARIS***

It is chronic inflammation of the skin with unknown cause. There are no essential histological features for diagnosis of prurigo nodularis. However, hyperkeratosis, parakeratosis, hypergranulosis, acanthosis, papillomatosis with papillary dermis showing vertically oriented collagen bundles are seen. Occasional cases show neural hyperplasia

which was also seen in 50% of the cases included in the present study. [10]

In both the cases of the present study, one was a 40 year old female and another one was 50 year old female. Both presented with intense itching. Age ranges from 31-50 years.Both the cases showed marked hyperkeratosis and acanthosis of epidermis [100%]. Dermis showed mild infiltration by lymphocytes and plasma cells. The characteristic feature seen in both these cases was hypertrophy and proliferation of dermal nerves [50%].

Mullins TB et al[91] in their study in 2024 showed that lesions of prurigo nodularis are intensely pruritic and have a diverse age group. These findings corroborated with the present study as the patients included in the present study also presented to the dermatologist with intense itching.

Alrabiah K et al[92] in their study on prurigo nodularis in 2023 showed that it may affect persons of any age, including children and elderly. The patients included in the present study were 40 years and 50 years old. This study also proposed that Substance P has an important role indermal neuronal hyperplasia which was in response to increased expression of nerve growth factor. Increased levels of substance P are also seen in these patients.

***MORPHEA***

Morphea is identified as thickened and fibrosed skin. It may present as localised or systemic forms. Lesions are seen as early inflammatory, intermediate and late sclerotic. Early lesions show more pronounced lymphoplasmacytic infiltrate in the reticular dermis with somewhat thickened collagen bundles. With time, this infiltrate involves the subcutaneous tissue. However, the late sclerotic stage shows minimal infiltrate with closely packed thickened collagen bundles.[10]

Of all 3 cases, 2 [67%] were females and one [33%] was male. Age ranges from 30-60 years.Of 3 cases of morphea, epidermis was insignificant with mild acanthosis seen in 1 case [33.3%]. Dermis showed thickened collagen bundles in 100% of the cases with

extension into the subcutaneous tissue. Mild infiltration of the papillary dermis by lymphoplasmacytic inflammatory infiltrate was seen in 1 case [33.3%]. Increased pigmentation in the basal layer of the epidermis was seen in 2 cases [66.6%].

A study conducted by Daniel walker et al[93] in 2017 showed that 80% females and 20% males were affected by morphea. The present study also showed female preponderance which was corroborated by another study conducted by Rodríguez-Salgado P et al. [93]

Choi S et al[95] in a study conducted in 2023 showed that histopathological featuresof morphea were seen as dermal and subcutaneous sclerosis along with plasma cell infiltration to be more prevalent in patients with Generalised Morphea than localised morphea or systemic sclerosis (p=0.046 and p=0.028, respectively). Pigment incontinence was also seen in few cases. These findings were in concordance with the histopathology seen in the present study.

***SUBACUTE SPONGIOTIC DERMATITIS***

Both the patients were males [100%] with one being a 34 year old and another being a 38 year old.Both the cases showed acanthotic and parakeratotic epidermis with mild to moderate spongiosis. Upper layers of the epidermis showed scattered lymphocytes, plasma cells and neutrophils. Dermis showed sparse perivascular lymphocytic inflammatory infiltrate.

Gru et al[96] in their study in 2017 on spongiotic dermatitis showed that histologically, the fluid accumulation in the intercellular spaces of the epidermis which is known as spongiosis along with parakeratosis in the epidermis are seen. Also seen were inflammatory cells extending into the epidermis, a term that is called exocytosis. Similar findings were seen in the present study.

A study conducted by Tanei R et al[97] in 2022 also showed similar histological findings in skin biopsies examined for spongiotic dermatitis.

***AMYLOIDOSIS***

In our study solitary case of cutaneous amyloidosis was 40 year old female patient came with a gradual increasing brownish papule since last one month associated with mild itching. Skin biopsy showed acanthotic epidermis with papillary dermis showing deposition of eosinophilic material. Congo red stain with apple green birefringence under polarized microscopy confirmed the deposited material to be amyloid.

Mehrotra K et al[98] in a study conducted in 2017 showed female preponderance in patients with primary cutaneous amyloidosis. This was corroborated with the present study in which there was only one case of a female patient. It further showed small, amorphous globules of eosinophilic material in papillary dermis along with pigmentary incontinence and epidermal changes like hyperkeratosis, acanthosis, hypergranulosis, parakeratosis and atrophy of epidermis in 80% cases, however, the present study showed 100% presence of hyaline eosinophilic material which was confirmed on congo red stain and immunofluorescence.

ŻychowskaMet al[99] in their study in 2021 focused more on lichen amyloidosis with histopathological examination displaying hyperkeratotic epidermis, mild inflammatory infiltrate composed of lymphocytes, histiocytes, and granulocytes in the upper dermis, and deposits of eosinophilic material in the papillary dermis, which stained positive with Congo red.

A study conducted in 2021 by Sinha Akanchha et al[100] showed female preponderance with histopathological examination small globular deposits of amorphous eosinophilic acellular material in the papillary dermis along with mild perivascular lymphocytic infiltration in 100% of the cases. Those cases that showed changes in the epidermis were identified as lichen amyloidosis and those that had an unremarkable epidermis were specified as macular amyloidosis.

***FIXED DRUG REACTION***

In the present study, a48 year old female patient presented to the dermatologist with blackish discolouration of the right arm since last 5-6 months associated with mild pain. Skin biopsy showed hyperkeratotic and parakeratotic epidermis with occasional scattered dyskeratotic keratinocytes in the epidermis. Prominent basal cell vacuolar degeneration noted. Dermis shows inflammatory infiltrate comprising of lymphocytes, histiocytes and occasional eosinophils around the blood vessels.

Weyers W et al[101] in a study conducted in 2011 proposed that an important criteria to diagnose drug eruptions was presence of eosinophils, which were also seen in the case included in the present study but the other authors dwelled upon the fact that presence of eosinophils does not make a diagnosis of drug reaction to be the correct diagnosis. However, the absence of eosinophils does not rule out a drug eruption also. The histopathologic descriptions of drug eruptions are indefinite and biopsy of drug eruptions are insignificant.

***SEBORRHOEIC KERATOSIS***

It is very common skin condition seen as single or multiple lesions on face, trunk and extremities. These lesions appear as ‘stuck on’ surface on the affected area.[10] We had 4 cases of seborrhoeic keratosis, 3 [75%] were seen in females and one case [25%] was seen in male. The age ranged from 55-75 years. These patients presented with gradually increasing blackish plaque over a period of 6 months to 1 year. All the 4 cases had past history of aggravation of symptoms of itching after exposure to sunlight. 100% of the cases showed hyperkeratosis, acanthosis and papillomatosis. Horn cysts were seen in 3 cases [75%]. The epidermis showed proliferation of basaloid cells in 100% of the cases along with squamous cell proliferation in most of the cases. Dermis showed moderate infiltration by lymphocytes and plasma cells in 100% of the cases.

Nam kyungRoh et al[31] in their study conducted in 2016 showed seborrhoeic keratosis to be seen in individuals with age greater than 50

years. These lesions were seen on sun exposed areas of the skin. These findings were corroborated with the present study. All the cases showed aggravation of the symptoms of itching after exposure to sunlight. All the cases had common histopathological features of hyperkeratosis, acanthosis and papillomatosis.

Ye et al[102] in their study conducted in 2021 showed that among all the patients with seborrhoeic keratosis, 49.1% were men and women accounted for 50.9% which is in concordance with the present study with female showing 75% prevalence while males showed 25% prevalence.

Shah et al[103] in a study conducted in 2018 showed hyperkeratosis (69.33%) being the most common histological feature of seborrhoeic keratosis followed by acanthosis (66.66%), papillomatosis (60%), pigmentation (34.66%), horn cyst (33.33%), orthokeratosis (32%), and parakeratosis (17.33%). However, the present study showed hyperkeratosis, acanthosis and papillomatosis in 100% of the cases followed by horn cysts in 75% cases. Basaloid cell proliferation was seen in all the cases with moderate dermal infiltration. The similar findings were seen in another study conducted by Geethu Francis Alapatt et al[104] where pigmentation(78%) was the most common followed by papillomatosis (68%), acanthosis (56%), hyperkeratosis (54%)and horn cyst formation (44%).

A study by Kitamura et al[105] showed that Seborrhoeic keratosis manifests a high degree of acanthosis and relatively abundant dilated vessels in the upper dermis.

***VERRUCA VULGARIS***

Verruca vulgaris, also known as the common wart, is the most common cutaneous manifestation of human papilloma viruses. They are mainly seen as painless lesions on fingers and hands. However, palms and soles can also be involved.[10]

Of all 4 cases of verruca vulgaris, 3 [75%] were females and one [25%] was male in the present study.The age distribution was from 31-50 years. These patients presented as hyperkeratotic papules over the

skin which developed slowly over period of 2-3 years. These patients were later found to be HPV positive. Of all the 4 cases, two had lesions over feet and other two had lesions on hands. On histopathological examination, all the 4 cases [100%] showed acanthosis, hyperkeratosis, papillomatosis of the epidermis, elongation of rate ridges and koilocytes in upper layers of epidermis.

Al Aboud AM et al[106] in their updated study on warts in 2022 showed that there is HPV association and common histopathological features seen are acanthosis, papillomatosis, hypergranulosis, elongated rete ridges pointing radially towards the center of the lesion. In the granular layer, cells infected with HPV have coarse keratohyalin granules and vacuoles surrounding wrinkled-appearing nuclei which are known as Koilocytic cells are seen. The similar kind of changes was seen in the present study with 100% HPV association.

A study conducted by Mustafa Alasiz[107] in 2021 proposed papillomatosis and koilocytosis as two most important histopathological features which were seen in 100% of the cases included in the present study.

***MELANOCYTIC NEVI***

The term 'Nevus' refers to benign neoplasms exhibiting melanocytic proliferation. They are rarely seen at birth and mostly manifest in early adulthood. But they may also be seen in middle aged adults which were concordant with the present study showing majority of the cases in 21-40 age group.

Histologically the melanocytic nevi are subcategorized into junctional, compound and intradermal nevi.[10] However, the cases included in the present study were of intradermal nevus, becker's nevus and verrucous epidermal nevus.

Of the 8 benign melanocytic hyperpigmented lesions comprising of intradermal nevus, verrucous epidermal nevus and becker's nevus, all the 8 cases [100%] showed hyperkeratosis, elongated rete ridges with perivascular inflammation. Nevus cell were seen in dermis in 4 cases [50%]. Four cases [50%] showed hypergranulosis. Orthokeratosis was

seen in 2 cases [25%] while papillomatosis was seen in one case [12.5%]. Three cases [37.5%] showed acanthosis and increasedbasal cell layer pigmentation. Vacuolated cells were seen in upper layers of epidermis in one case [12.5%]. No junctional activity/ significant mitosis/ nuclear atypia was seen in any of the cases examined.

The most common melanocytic nevi was intradermal nevus which accounted for 50% of total melanocytic nevi. Similar observations were made by Pailoor K et al[43] and Abhishek Singh et al[67] in which intradermal nevus accounted for 66% and 69% of the cases respectively. In a study done by Gundalli Set al[11], 84.6% of cases were of intradermal nevus.

A study conducted in 2019 by Nilgunsogutcu et al[108] showed histological features of intradermal nevi as nevus cell in dermis (100%) with some intradermal nevi showing signs of hyperkeratosis and papillomatosis resemble seborrhoeic keratosis.

GundalliSet a[11] in their study conducted in 2016 on various melanocytic lesions showed nevus cells confined to the dermis in 100% of the cases where they were arranged in nests and cords with no junctional activity in case of intradermal nevi. The present study also showed these characteristic findings of intradermal nevi in 100% of the cases.

Becker's melanosis presents as solitary hyperpigmented hypertrichotic patch on shoulder, back or chest in adult male. It may also be seen as multiple lesions in women too. [10]

Ankita Patil et al[109] in a study conducted in 2023 showed histological features of Becker's nevus as absence of melanocytic nevus cells which is the diagnostic feature along with hyperkeratosis in 71.4% cases, acanthosis in 91.4% cases, elongation of rete ridges in 91.4% cases and basal layer pigmentation 100%cases. Smooth muscle hyperplasia was observed in 11.4% cases. These findings were similar to the present study.

Abdulwahab RA et al[110] in their case study conducted in 2023 on becker's nevus showed histopathological features in all 100% of the cases as epidermal hyperkeratosis, acanthosis, and focal regular

elongation of rete ridges with clubbing. There was increased basal layer pigmentation. The dermis revealed focal areas with pigment incontinence. There was no increase in smooth muscle in the dermis or hyperplasia of the hair follicles which was in concordance with the present study.

Verrucous epidermal nevus is a variant of epidermal nevi and presents as intensely pruritic persistent lesion. It may be seen in children or adults. Majority of the lesions are seen on lower limbs, however, vulvar and perianal areas can also be involved. [10]

Anna Elisa verzi et al[111] in their case study conducted on 8 cases of verrucous epidermal nevus in 2019 showed diagnosis being made mainly on clinical presentation with histopathological examination revealing epidermal hyperplasia, papillomatosis with elongation of rete ridges, and pigmented keratinocytes surrounding the dermal papillae. Correlation between the clinical and histopathological findings led to diagnosis in the present study.

***MALIGNANT LESIONS***

In the hyperpigmented malignant lesions, most common entity was of Basal cell carcinoma constituting 67% of all malignant lesions followed by 33% of malignant melanoma which is similar to the study of Ganesh Basina et al[64] where the incidence of BCC was 80%.

In the present study, it was observed that the malignant hyperpigmented skin lesions were common in the age group of 51-80 years. This corroborated with the study done by Ganesh Basina et al[64] of 60 cases which showed peak age incidence of malignant lesions in 51-60 years. Goswami et al[73] in his study showed majority of the patients with malignant skin conditions were >60 years in age.

In our present study the most common site of presentation of malignant hyperpigmented lesions was head and neck (face), accounting for 83.3% of the cases. Similar observations were made by Laishram RS et al[112] (51.4%) and Crasta J et al[113] (70%).

Basal cell carcinoma is considered to be the most common skin cancer in humans. Most common disposing factor for BCC is prolonged

exposure to sunlight. It is a locally aggressive tumor, and by the rule, does not metastasize. However, literature shows incidence of metastasis in 0.01% cases. Most of the patients present with a large ulcerated lesion on hair bearing area, most commonly on the face with evidence of recurrence.

The clinical subtypes of BCC include: Superficial, nodular, micronodular, infiltrating and fibroepithelioma.[10]Nodular variety is its most common variety as seen by McDaniel B et al[61] in their updated study in 2024.

Of the 4 cases of malignantlesions of basal cell carcinoma, all the cases showed orthokeratosis, nuclear pleomorphism, atypical basaloid cells, increased basal cell pigmentation and perivascular inflammation [100%]. Out of total 4 cases, hyperkeratosis and parakeratosis was seen in 3 cases [75%]. All the four cases were diagnosed as nodular variant of basal cell carcinoma as the lesions were relatively circumscribed masses clinically and were seen in the head and neck area. Pigment incontinence was also seen which is also a feature of nodular variant.

These similar results were also seen by Balakrishanan S[114] in the study conducted in 2022 describing histology of various variants of basal cell carcinoma where classical BCC was described as a basaloid epithelial tumor arising from the epidermis. Peripheral palisading was seen along with artefactual retraction clefts.The tumor showed centrally located nuclei, which was crowded with scatteredmitotic figuresand necrotic bodies. The similar histological features were seen in 100% cases of basal cell carcinoma in the present study.

Daniela- Elena Gheoca Mutu et al[115] in their study conducted on malignant melanoma in 2022 showed that mean age of the patients was 57.9±15.4 years with 90.9% cases in males. This is in concordance with the present study in which both the patients of malignant melanoma were males with their age being 57 years and 71 years respectively. The lesions were present over foot and leg in both the cases. Skin biopsy showed foci of epidermal ulceration in one case with dense inflammatory granulation, acute and chronic. The adjacent

epidermis in both the cases showed hyperkeratosis, parakeratosis and acanthosis. Dermis showed a tumor with tumor cells arranged in nests. Melanin pigment was seen around these nests and within cytoplasm of few cells. Few of the cells show prominent nucleoli as well. One case showed tumor cells extending upto the reticular dermis sparing the subcutaneous tissue giving Clark's classification for melanoma as level IV. Another case showed tumor cells only in the papillary dermis giving Clark's classification for melanoma as level II.

In the present study, positive correlation between clinical diagnosis and histopathological diagnosis was seen in 63 (70%) of the cases and negative correlation in 27(30%) of cases, thus emphasizing the need of histopathology in arriving at a conclusive diagnosis. Higher percentage of clinical concordance can be attributed to many differential diagnosis given by the dermatologist. Ganesh Basina et al[64] their study revealed positive correlation in 81% of cases and negative correlation in 19% of cases. Shushan SJ et al [63] in their study showed concordance of 78.9% and discordance of 21.1%. MruthyunjayappaS et al [30] in their study of 200 hyperpigmented skin lesions showed clinical concordance of 95% and discordance in 5%.

| CLINICOPATHOLOGICAL CORRELATION | Ganesh Basina et al[64] (n=60) | Shushan SJ et al[63] (n=85) | Mruthyunjaya ppaS et al[30] (n=200) | Present study (n=90) |
|---|---|---|---|---|
| CONCORDANCE | 49(81%) | 67(78.9%) | 190(95%) | 63(70%) |
| DISCORDANCE | 11(19%) | 18(21.1%) | 10(5%) | 27(30%) |
| TOTAL | 60(100%) | 85(100%) | 200(100%) | 90(100%) |

# SUMMARY AND CONCLUSION

The present study was conducted on 90 clinically diagnosed hyperpigmented skin biopsies that were received in the department of pathology, Dayanand Medical College and Hospital, Ludhiana over a period of 1.5 years (18 months) from 15 September 2022 to 14 March 2024.

The results and their interpretation are as under-

- Majority of the cases [28 cases,31.1%] of hyperpigmented skin lesions presented in third decade followed by fifth decade [16 cases,17.7%]. The mean age being 44.46 year.
- Benign lesions were more common in the fourth decade [33.3%] while malignant lesions were common after the fifth decade [100%].
- Female preponderance was noted with 54 cases in females [60%] and 36 cases in males [40%]. The ratio of male: female is 1:1.5
- Most common site of hyperpigmented lesions were seen in limbs [n=43,47.8%], followed by lesions over the trunk and back [n=20,22.2%].
- Majority of the lesions clinically presented as plaques [n=38,42.2%] followed by papules [n=18,20.0%], patches [n=16,17.7%], macules [n=8,8.8%], nodules [n=5,5.5%], ulcers [n=3,3.3%] and reticular pattern [n=2,2.2%].
- Bilateral involvementwas more common as compared to unilateral involvement. Generalized involvement of the body was least common.
- Majority of the cases presented as multiple lesions (73%) on the body while 27% presented as solitary lesion on various sites over the body.
- Majority (n=22,24.4%) of the cases came with the clinical diagnosis of lichen planus followed by discoid lupus erythematosus (n=13,14.4%), prurigo nodularis (n=10,11.1%), lichen planus pigmentosus (n=9,10.0%), dermatitis herpetiformis (n=8,8.8%), basal cell carcinoma (n=5,5.5%), lichen planus hypertrophicus (n=4,4.4%), intradermal nevus (n=4,4.4%), morphea (n=3,3.3%),

endogenous eczema (n=3, 3.3%), becker's nevus (n=2, 2.2%), livedo reticularis (n=2, 2.2%), granuloma annulare (n=2, 2.2%) and malignant melanoma (n=1, 1.1%).

- Majority (n=29,32.2%) of the cases were diagnosed as lichen planus and its variants followed by lichen simplex chronicus (n=10, 11.1%), discoid lupus erythematosus (n=8,8.8%), lichenoid drug eruption (n=7, 7.7%), postinflammatory hyperpigmentation (n=5,5.5%), seborrhoeic keratosis (n=4, 4.4%), verruca vulgaris (n=4,4.4%), intradermal nevus (n=4, 4.4%), basal cell carcinoma (n=4, 4.4%), becker's nevus (n=3, 3.3%), morphea (n=3, 3.3%), prurigo nodularis (n=2,2.2%), subacute spongiotic dermatitis (n=2,2.2%), malignant melanoma (n=2,2.2%), lichen amyloidosis (n=1,1.1%), fixed drug eruption (n=1,1.1%) and verrucous epidermal nevus (n=1,1.1%).
- Various types of lichen planus encountered in our study were classical lichen planus (n=15,51.7%) followed by lichen planopilaris (n=9,31.0%), lichen planus hypertrophicus (n=4, 13.7%) and lichen planus pigmentosus (n=1, 3.4%).
- Of the 16 benign tumors, there were 4 cases (25%) each of seborrhoeic keratosis, verruca vulgaris and intradermal nevus. There were 3 cases (18.75%) of becker's nevus and one case (6.25%) of verrucous epidermal nevus.
- Of the 6 malignant cases, 4 cases [66.6%] were of basal cell carcinoma and 2 cases [33.3%] were of malignant melanoma.
- Of the 90 hyperpigmented cases, 63 cases (70%) showed histopathological correlation with the clinical diagnosis while 27 cases (30%) were diagnosed only on histopathological examination.
- In conclusion, hyperpigmented lesions of the skin can have a variable etiology ranging from infectious diseases to malignant tumors. The exact diagnosis can only be achieved by a thorough correlation between the pathologist and the dermatologist.

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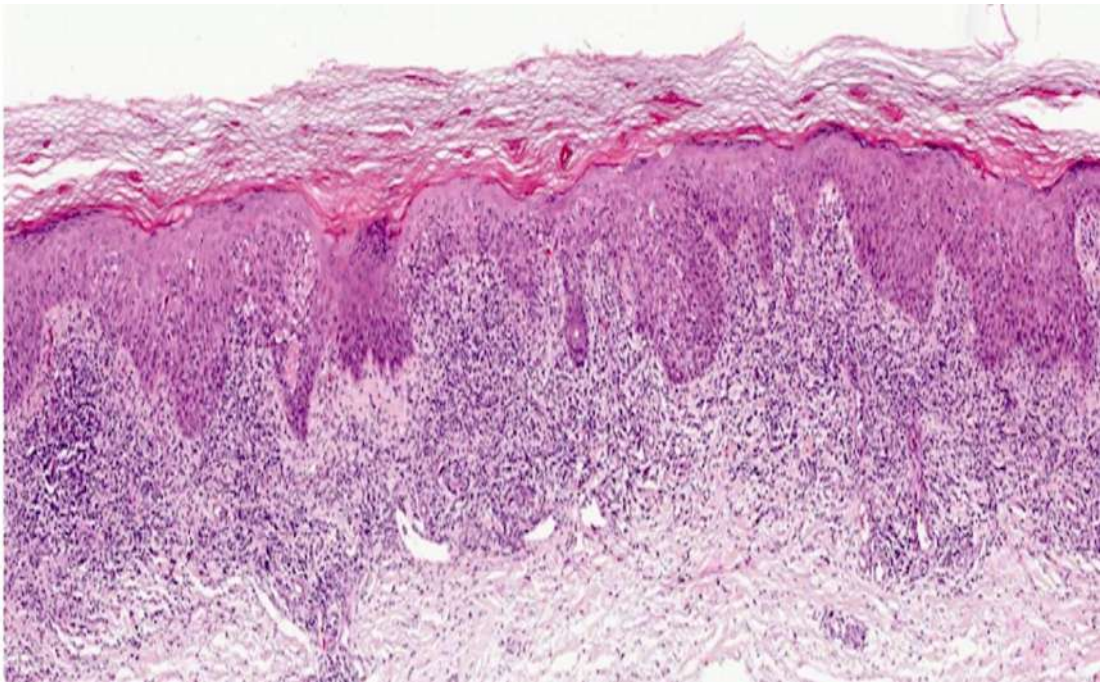
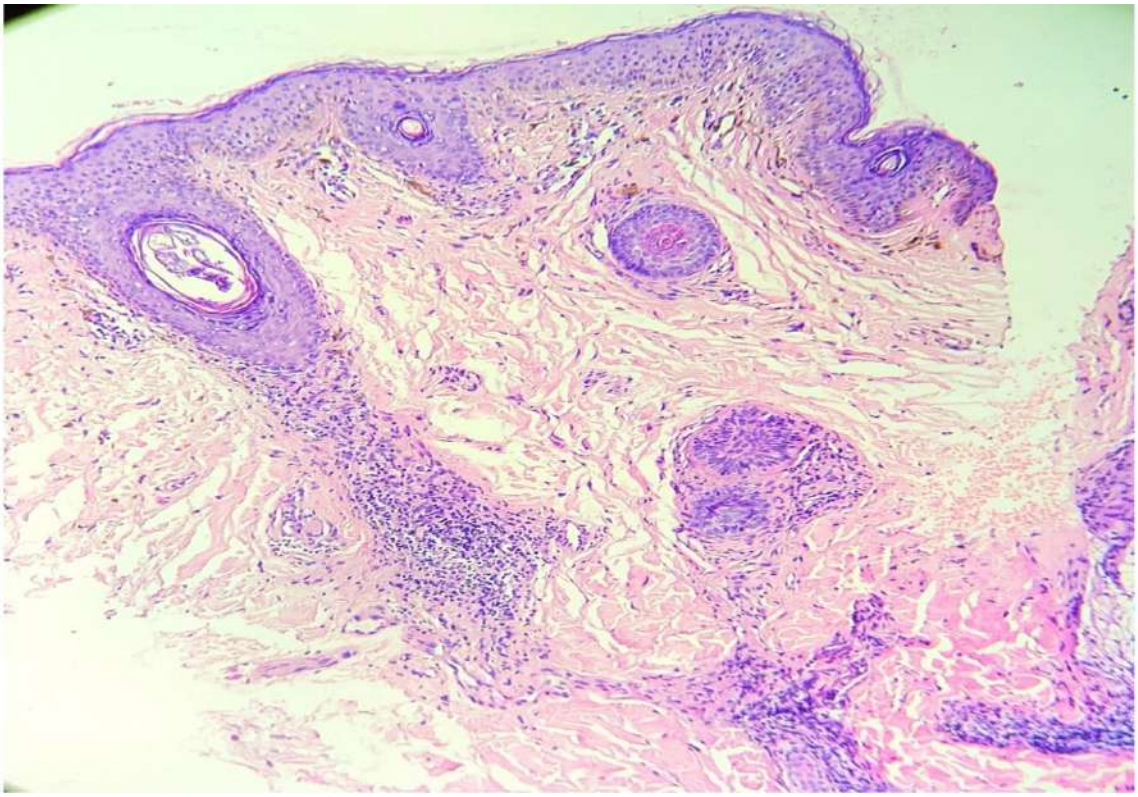
Picture 1: Photomicrograph showing clinically diagnosed case of lichen planus presenting with hyperpigmented macules over the arms

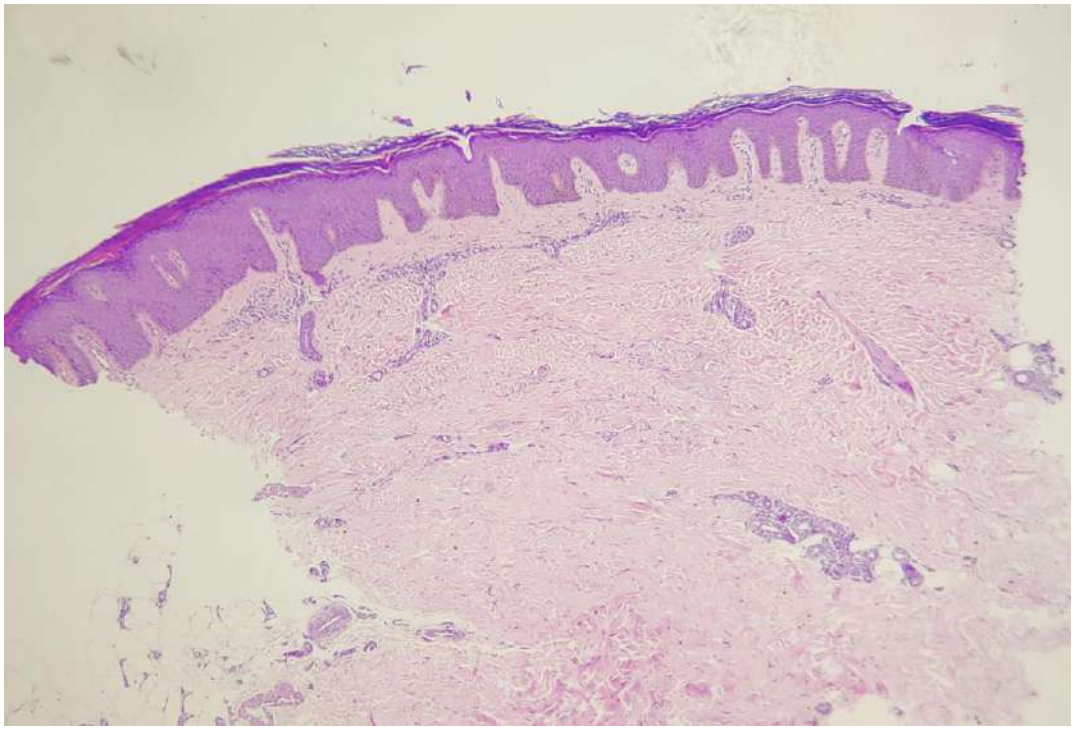


Picture 2: Lichen Planus- Photomicrograph shows band like lymphohistiocytic infiltrate at dermoepidermal junction with saw toothing of rete ridges and hyperkeratosis. [H&E, 10X]

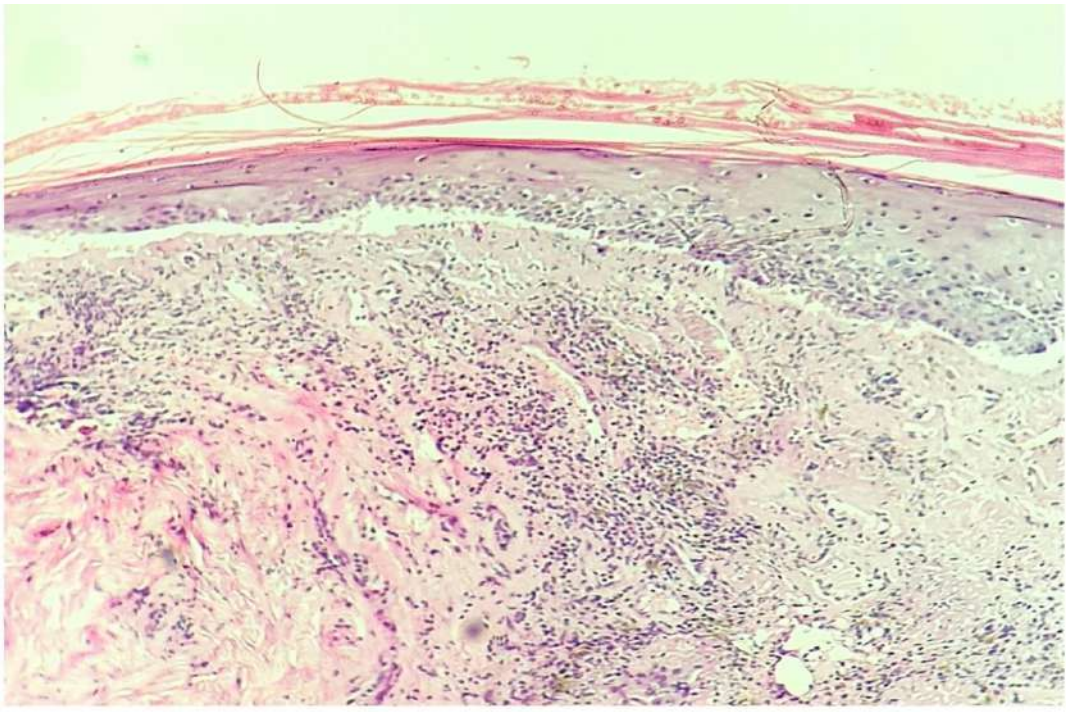


Picture 3: Lichen Planopilaris- Photomicrograph shows perifollicular lymphohistiocytic inflammatory infiltrate with pigment incontinence [H&E, 20X]

Picture 4: Lichen planus hypertrophicus: Photomicrograph shows wedge shaped hypergranulosis, hyperkeratosis and acanthosis with lymphohistiocytic inflammatopry infiltrate at base of rete ridges. [H&E, 20X]

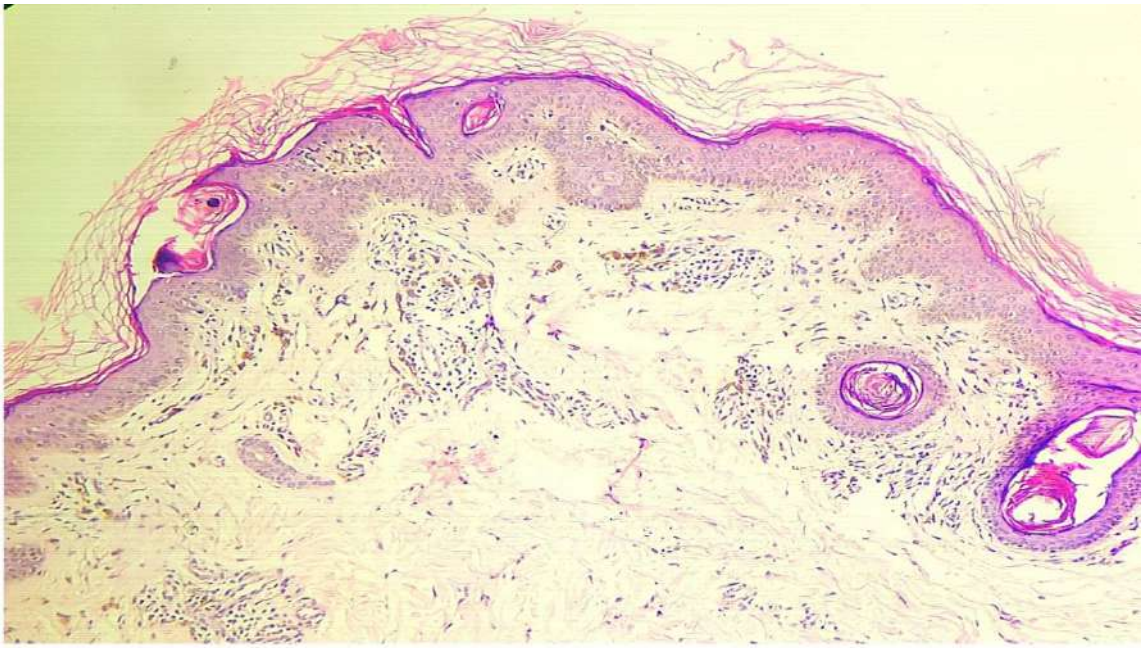


Picture 5: Lichen simplex chronicus: Photomicrograph shows hyperkeratosis, acanthosis, hypergranulosis and mild perivascular inflammatory infiltrate. Vertically oriented papillary dermal fibrosis is characteristically seen. [H&E, 20X]

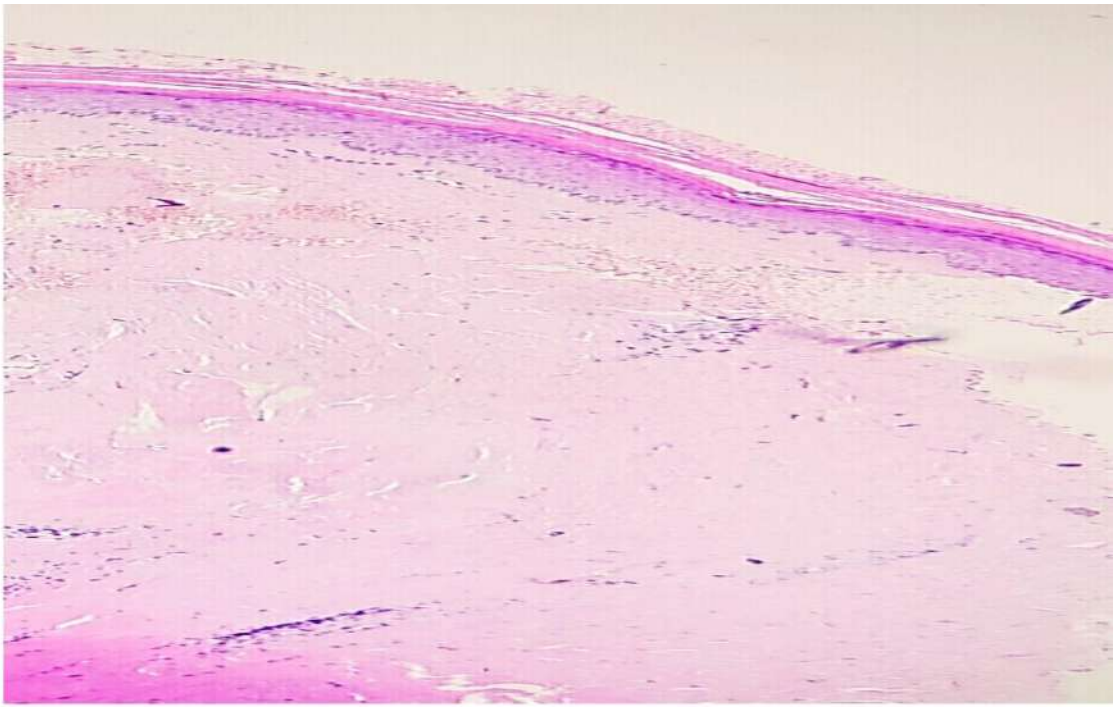


Picture 6: Discoid lupus erythematosus- Photomicrograph shows basal cell vacuolar degeneration with atrophy of epidermis and chronic inflammatory infiltrate in papillary dermis. [H&E, 400X]

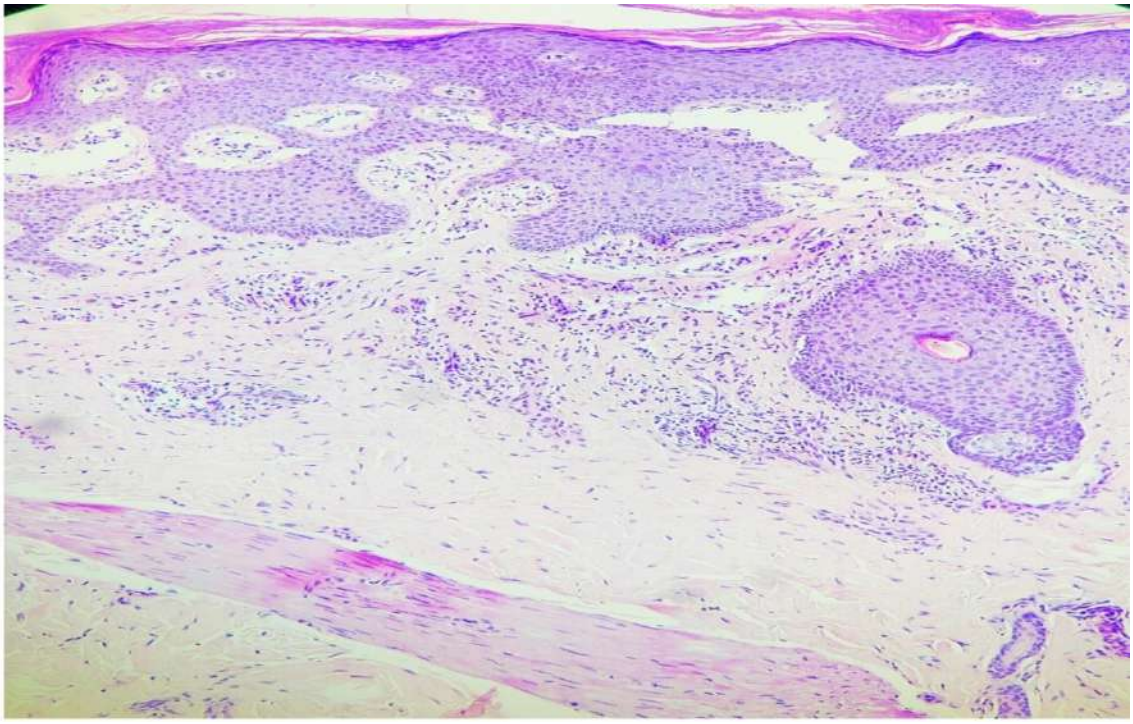
Picture 7: Post inflammatory hyperpigmentation- Photomicrograph showing hyperpigmented papulonodular lesions over back



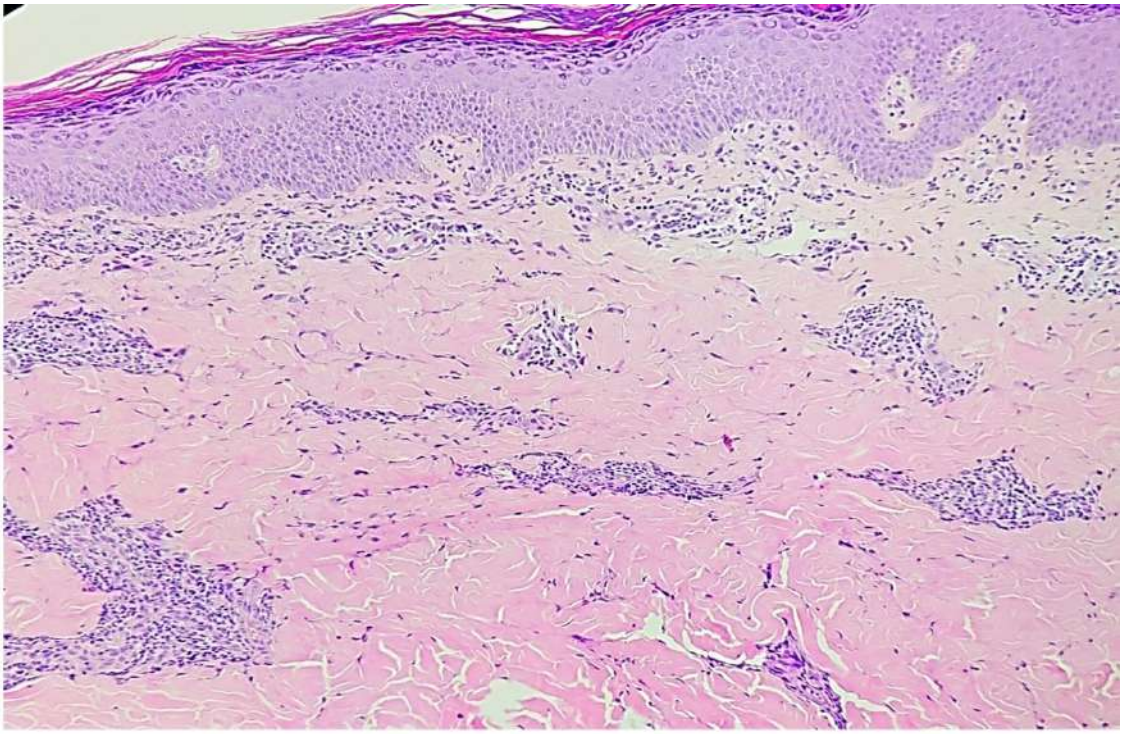
Picture 8: Postinflammatory pigmentation- Photomicrograph shows orthokeratotic epidermis. Increased pigmentation of the basal cell layer with pigment incontinence and increased fibrosis in the dermis seen. [H&E, 20X]



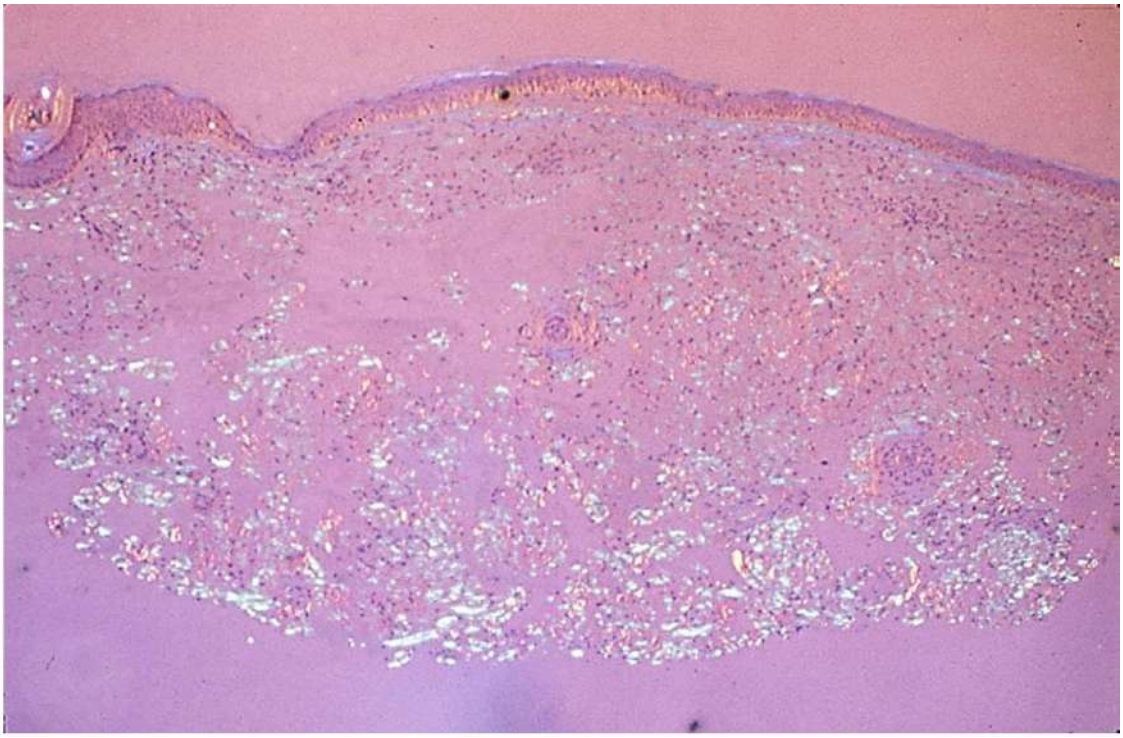
Picture 9: Morphea- Photomicrograph shows hyperkeratosis with increased basal cell pigmentation. Increased dermal collagenization seen. [H&E, 10X]



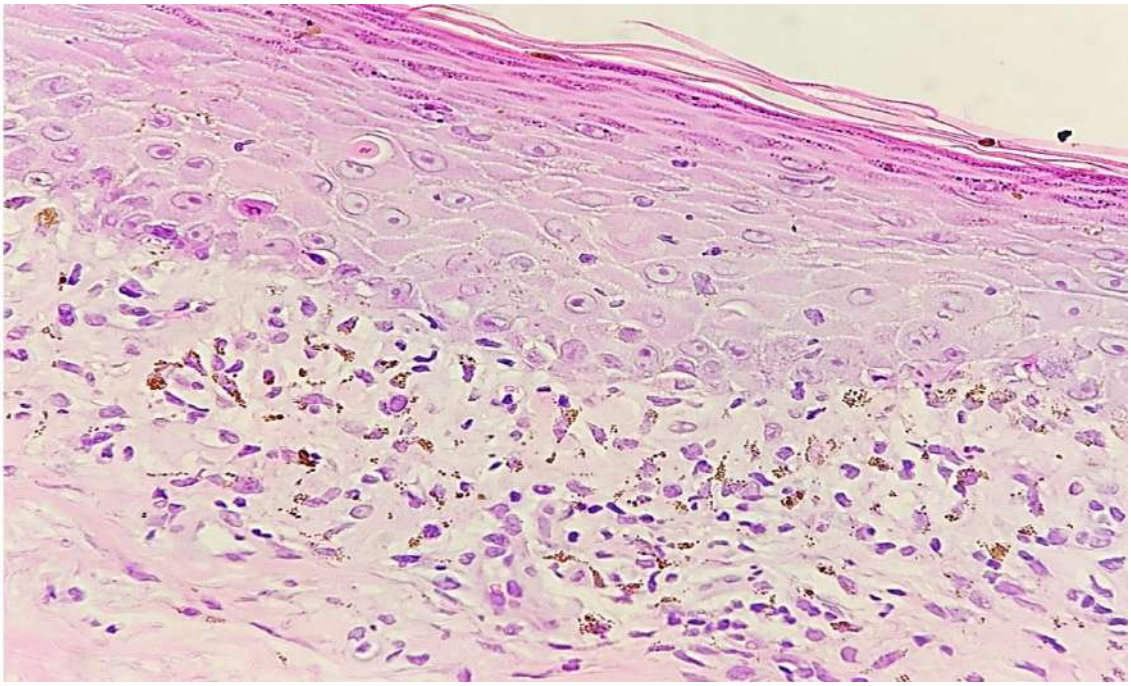
Picture 10: Prurigo nodularis- Photomicrograph shows hyperkeratosis, acanthosis of epidermis. Dermis shows hypertrophy of dermal nerves with infiltration of papillary dermis by lymphocytes, plasma cells and histiocytes. [H&E, 10X]



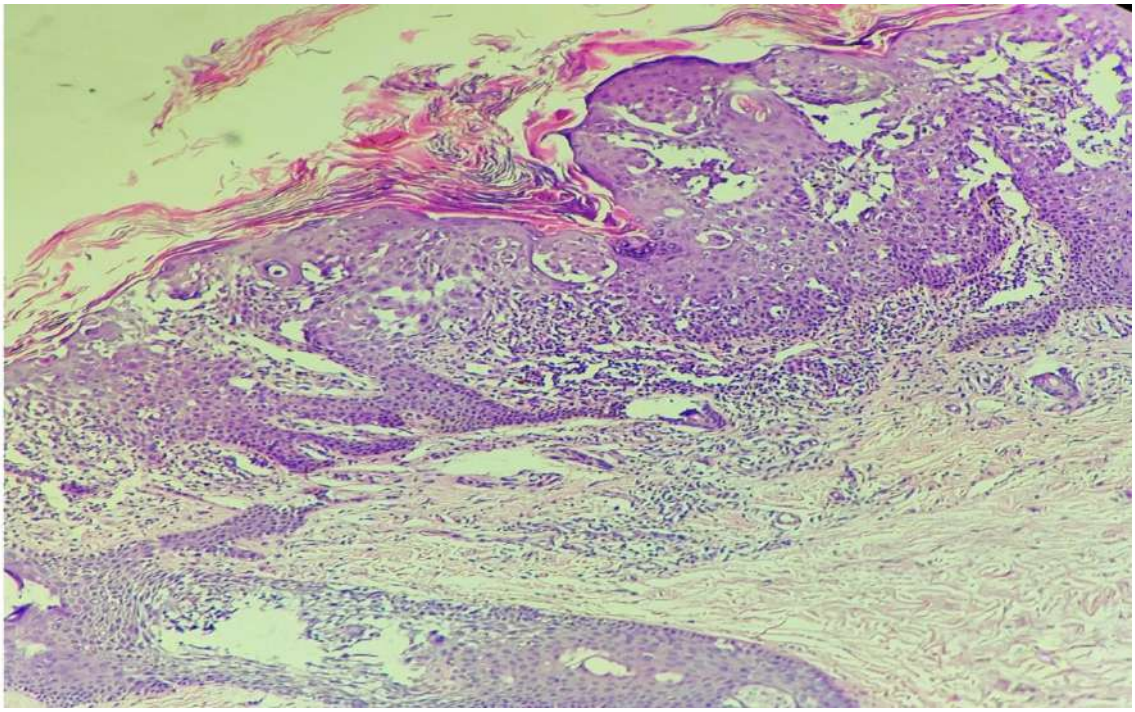
Picture 11: Amyloidosis- Photomicrograph shows eosinophilic material in the papillary dermis with acanthotic epidermis. [H&E, 10X]



Picture 12: Amyloidosis: Congo Red stain shows apple green birefringence under polarized light. [Congo Red, 10X]

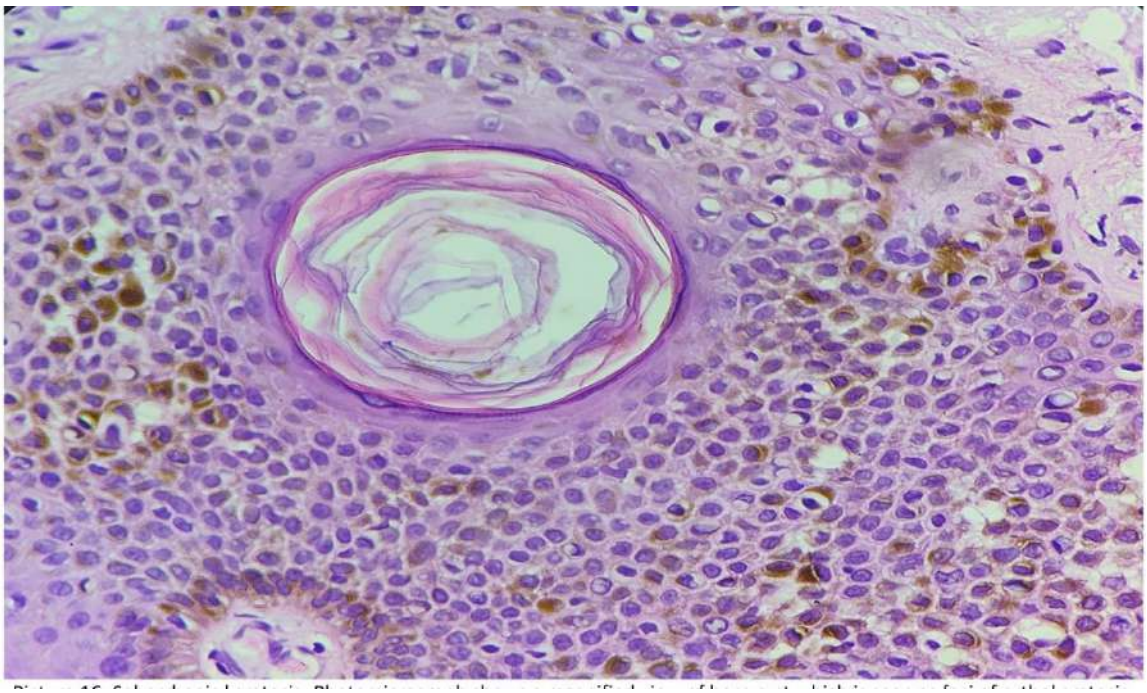


Picture 13: Lichenoid drug eruption- Photomicrograph shows hyperkeratosis, acanthosis with basal cell vacuolar degeneration. Dermis shows lymphoplasmacytic inflammatory infiltrate with few eosinophils at dermoepidermal junction with pigment incontinence. [H&E, 20X]

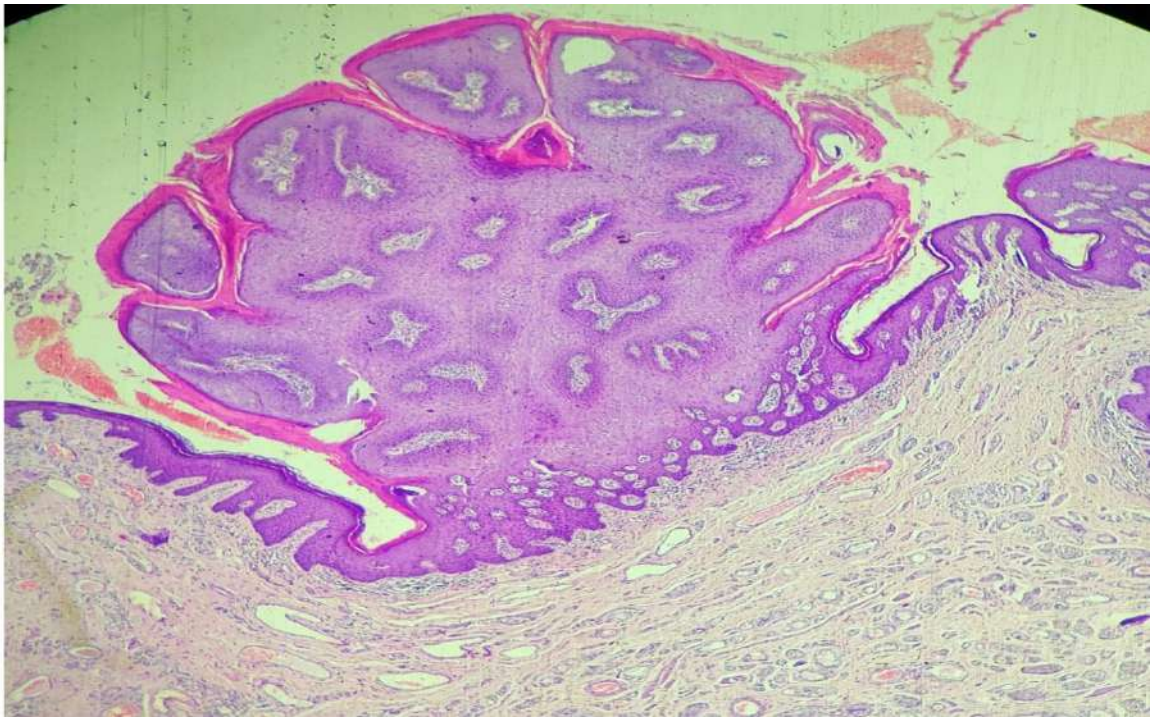


Picture 14: Subacute spongiotic dermatitis- Photomicrograph shows hyperkeratotic and acanthotic epidermis with spongiosis. Dermis showed moderate perivascular lymphohistiocytic inflammatory infiltrate. [H&E, 20X]

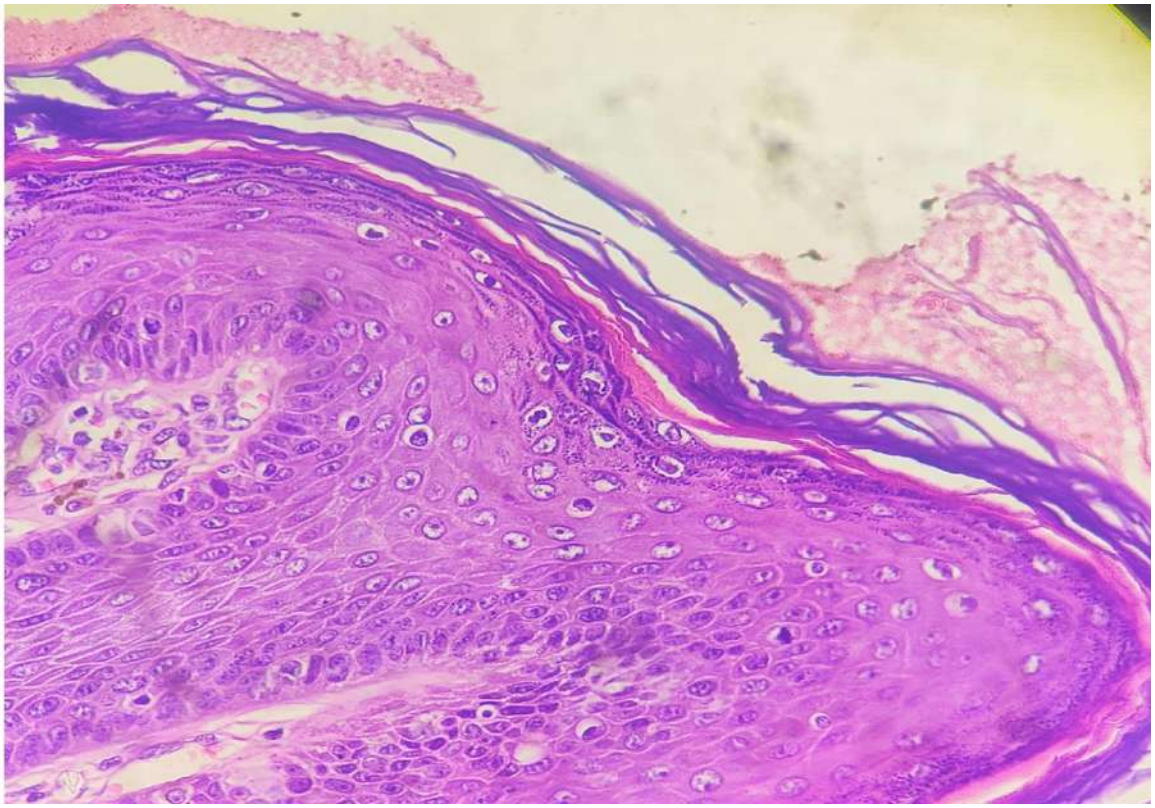
Picture 15: Seborrhoeic keratosis- Photomicrograph shows multiple horn cysts with proliferation of basaloid cells. [H&E, 100X]



Picture 16: Seborrhoeic keratosis- Photomicrograph shows a magnified view of horn cyst along with proliferation of basaloid cells. [H&E, 20X]

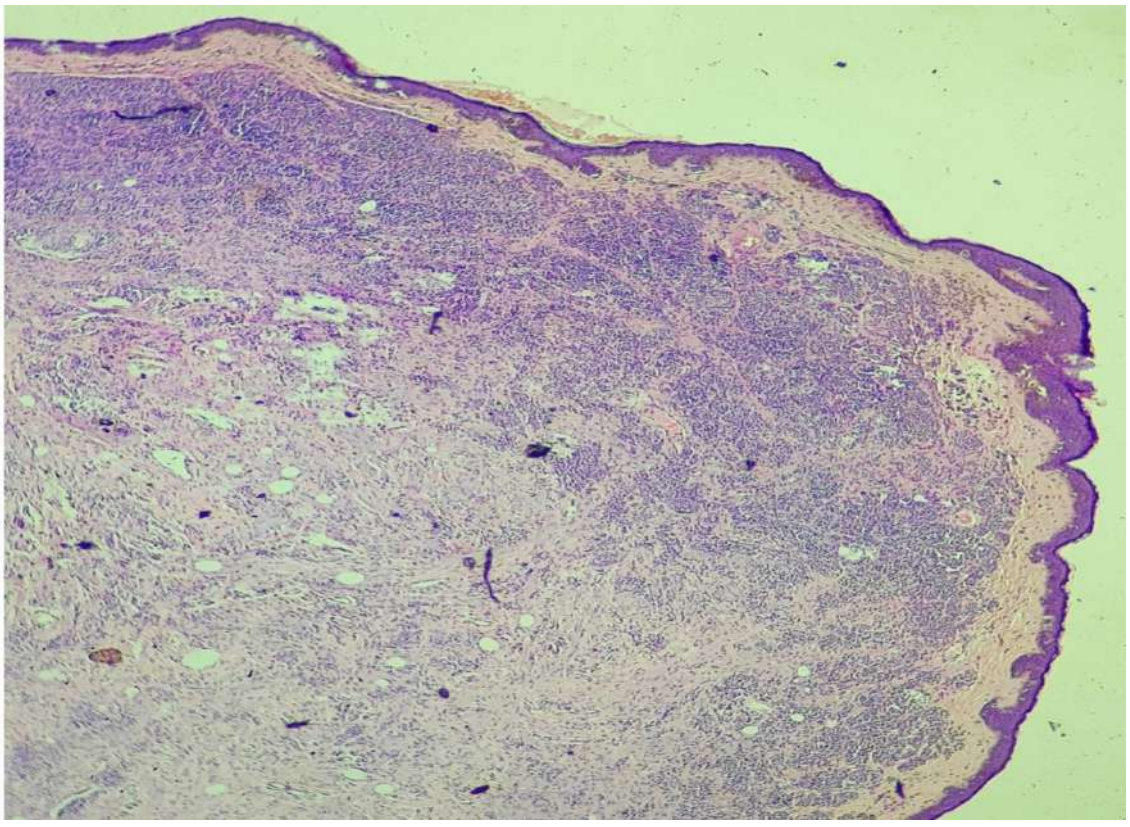


Picture 17: Verruca Vulgaris- Photomicrograph shows hyperkeratosis, papillomatosis and acanthosis of epidermis. [H&E, 10x]

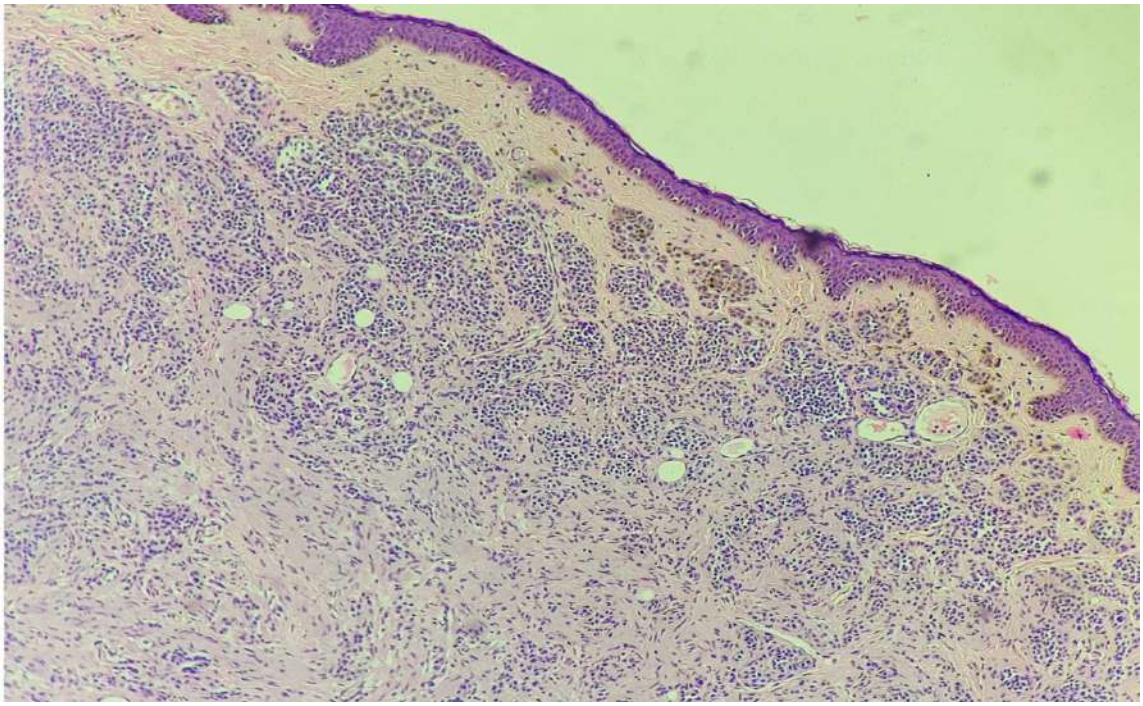


Picture 18: Verruca vulgaris- Photomicrograph shows koilocytes with resinoid nucleus and perinuclear halo. [H&E, 400X]

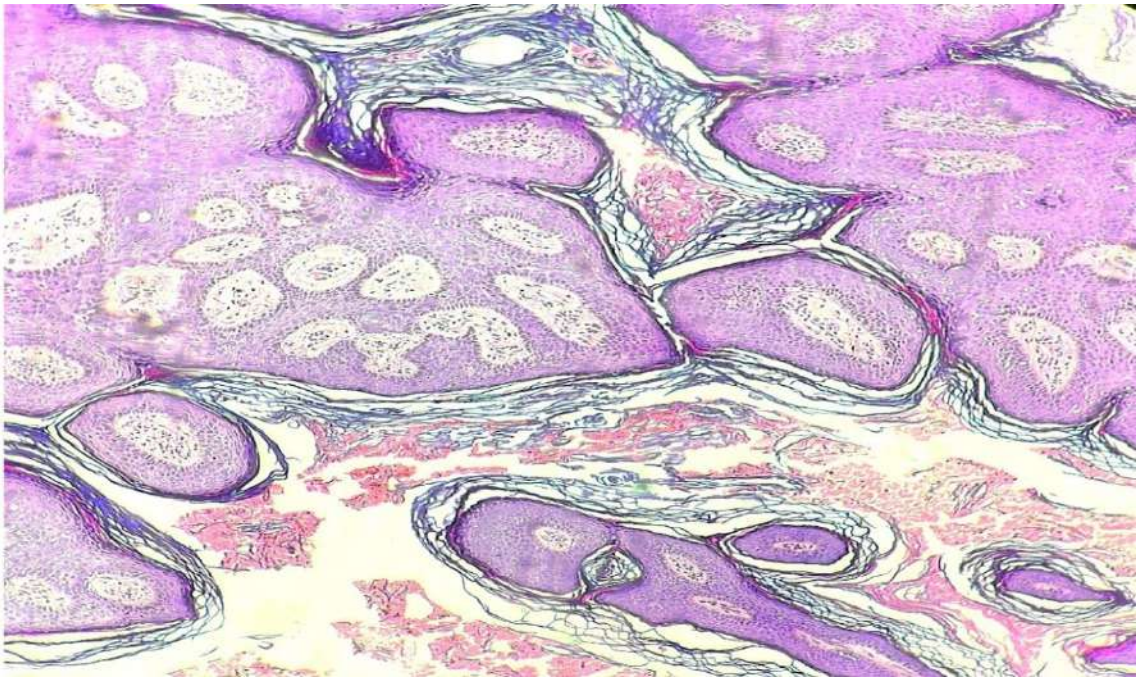
Picture 19: Intradermal nevus- Photomicrograph showing hyperpigmented nodule over the back



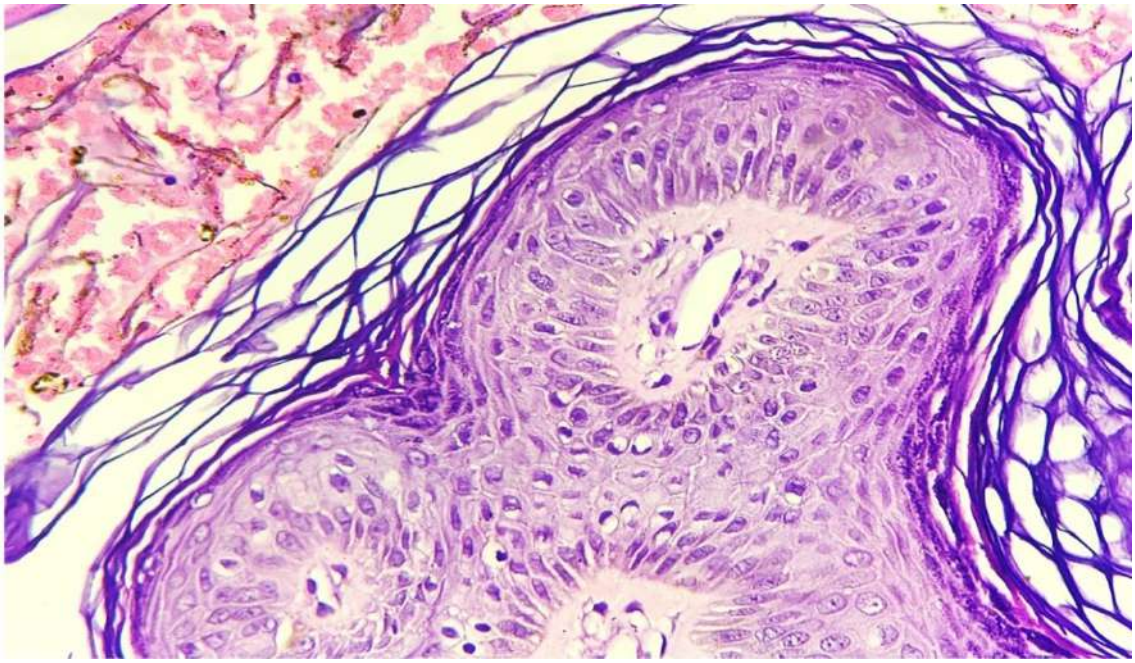
Picture 20: Intradermal nevus- Photomicrograph shows presence of nevus cells in the dermis with unremarkable epidermis. [H&E, 10X]



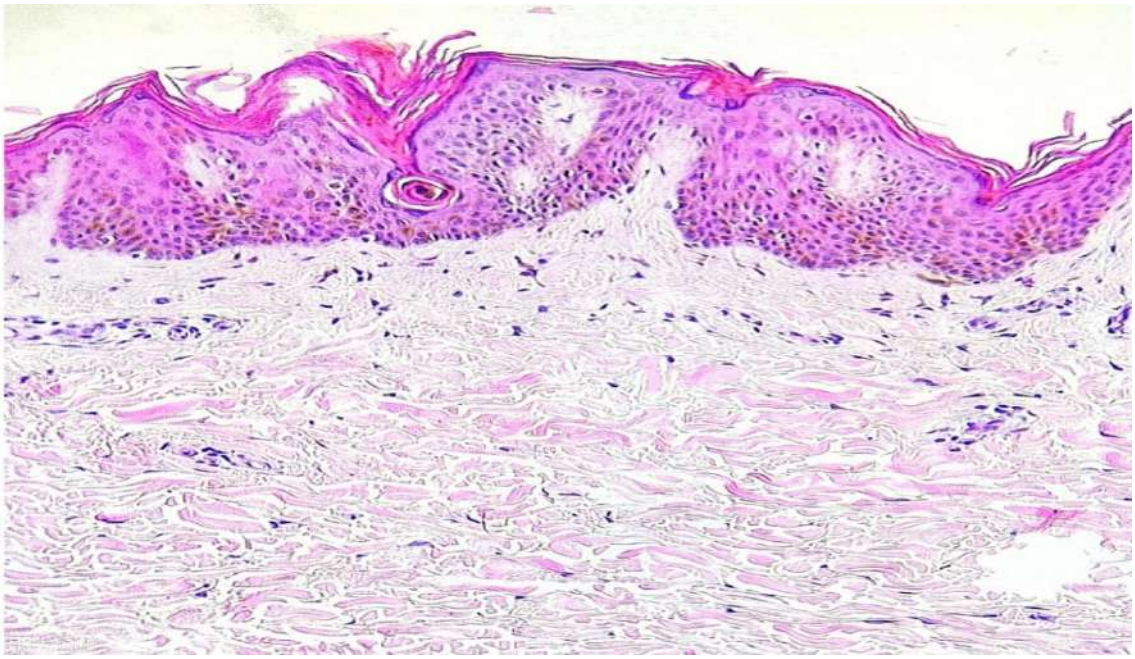
Picture 21: Intradermal nevus- Photomicrograph shows nevus cells with pigment incontinence in the dermis. [H&E,100X]



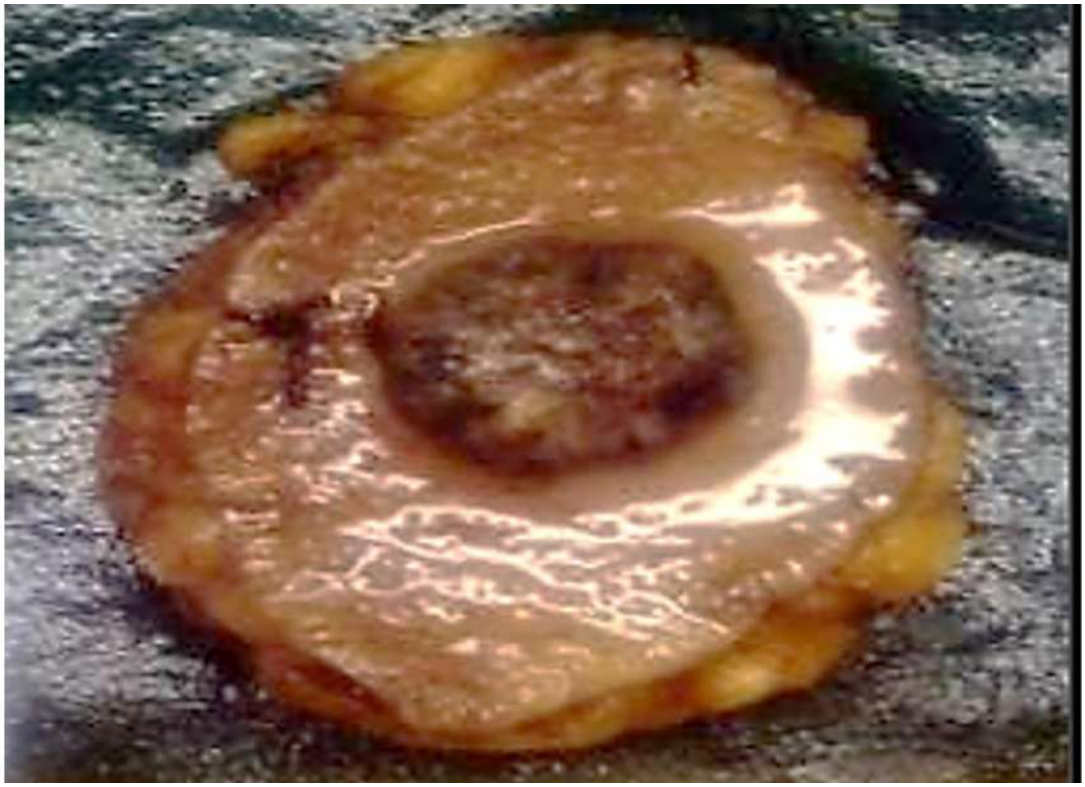
Picture 22: Verrucous epidermal nevus: Photomicrograph shows hyperkeratosis with pigmented keratinocytes lining the dermal papillae. [H&E, 100X]



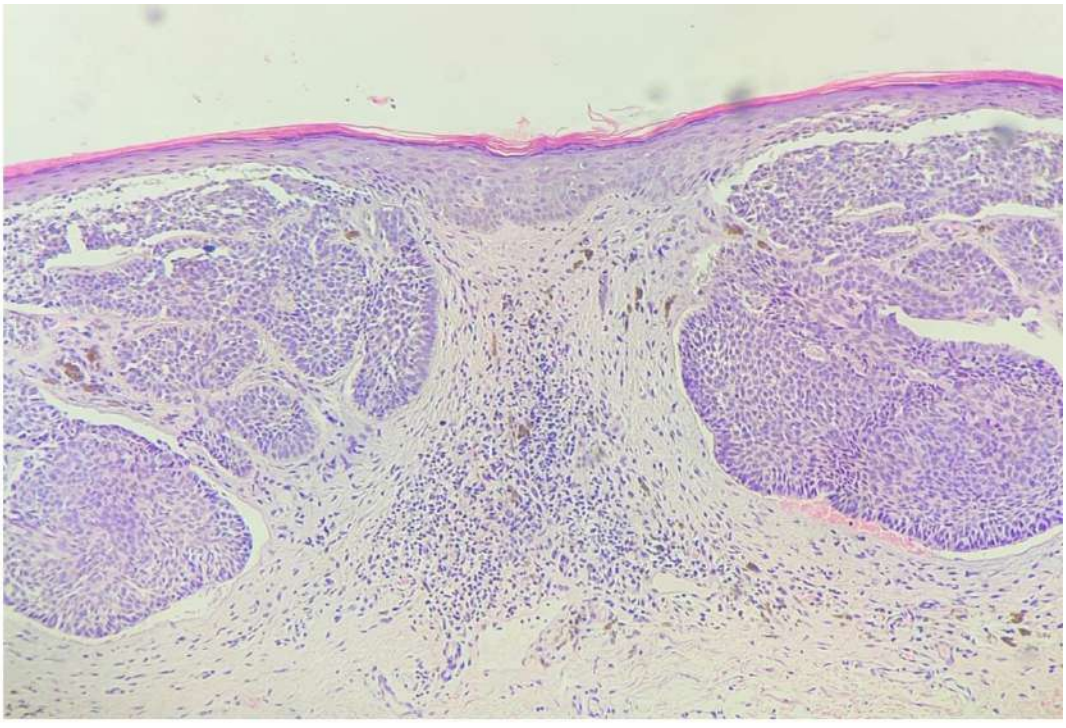
Picture 23: Verrucous epidermal nevus- Photomicrograph shows hypergranulosis with increased basal cell pigmentation. Pigmented keratinocytes are lining the dermal papillae. [H&E, 20X]



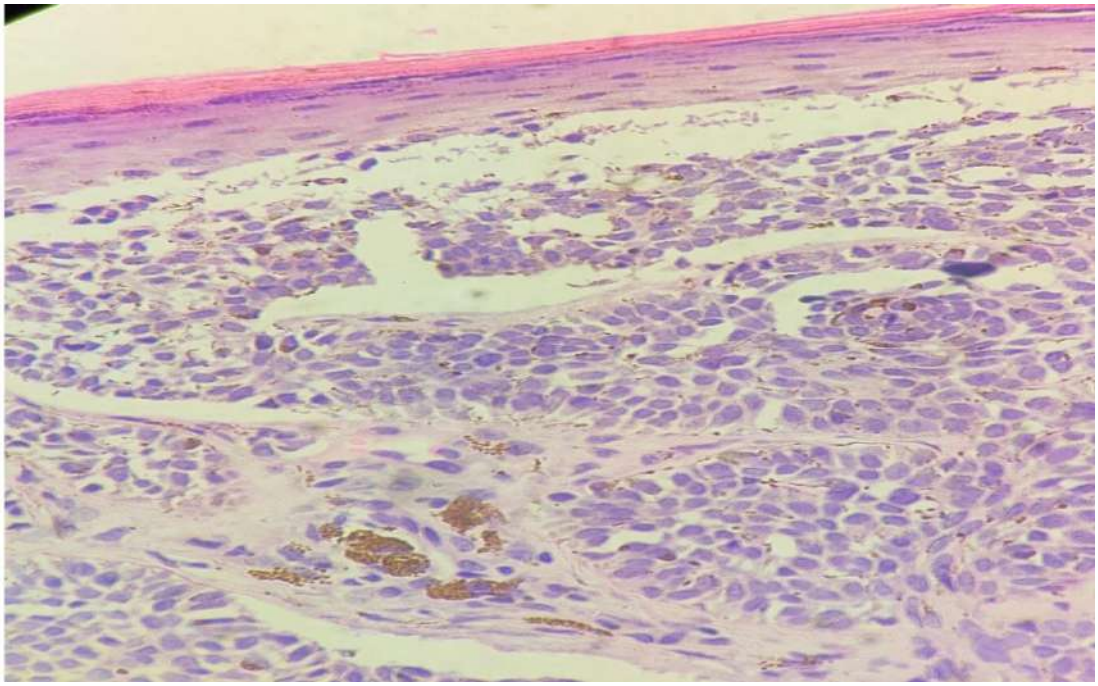
Picture 24: Becker's nevus- Photomicrograph shows hyperkeratosis, acanthosis with increased basal cell layer pigmentation. [H&E, 10X]



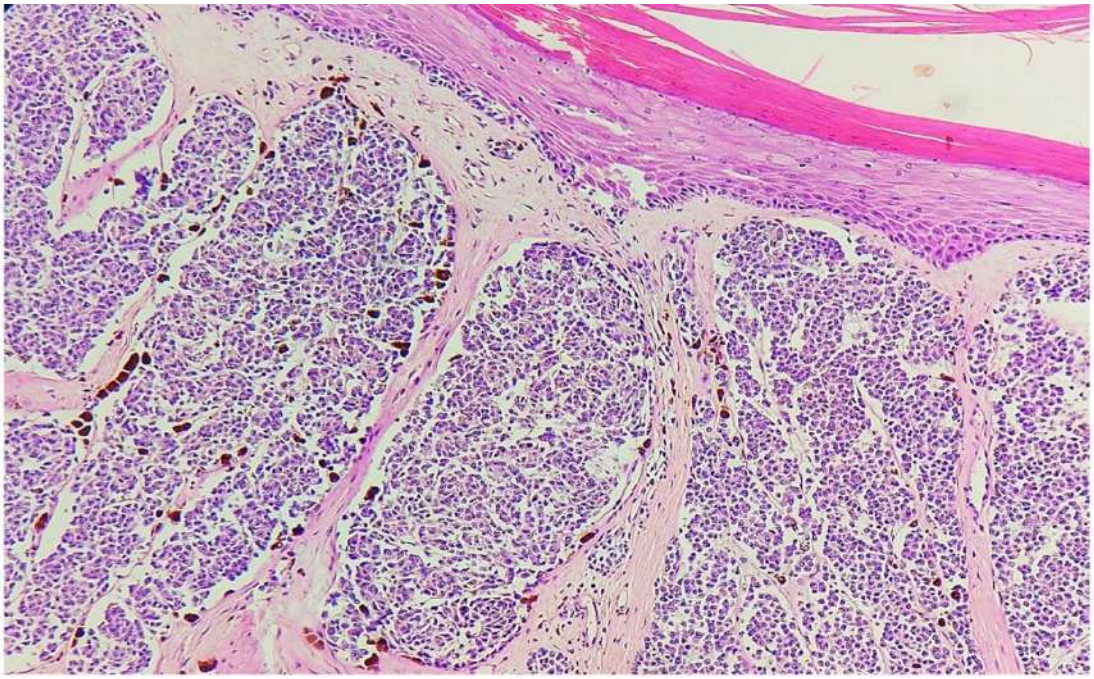
Picture 25: Basal cell carcinoma: Photomicrograph showing ulcerated area over the skin



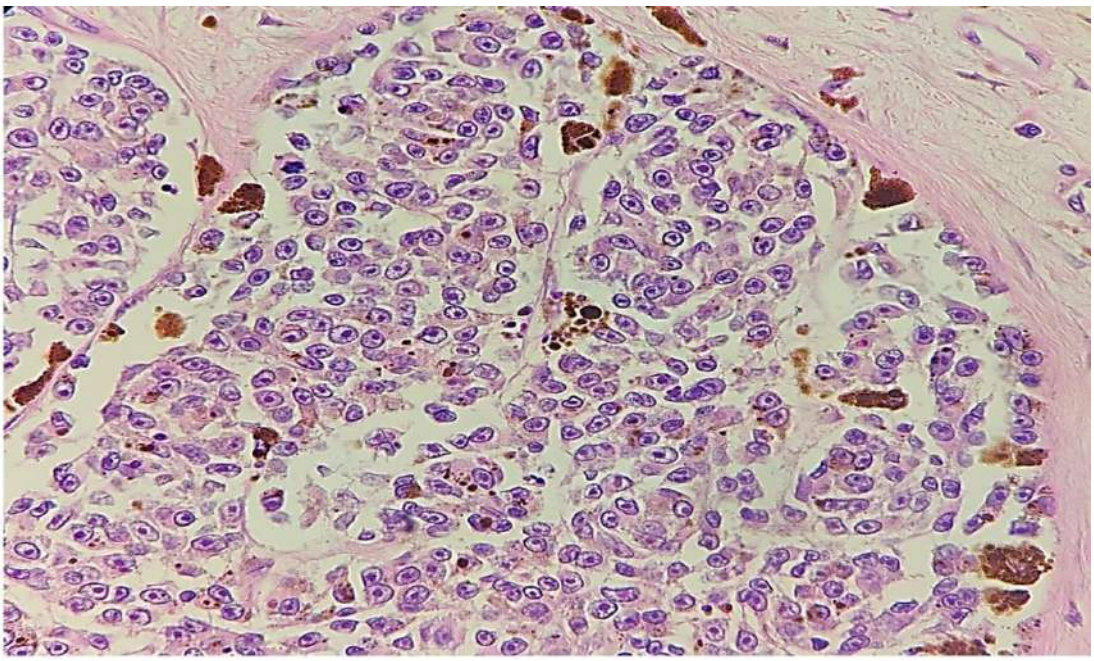
Picture 26: Basal cell carcinoma- Photomicrograph shows hyperkeratotic epidermis with nests of atypical basaloid cells in the dermis. Pigment incontinence seen. [H&E, 10X]



Picture 27: Basal cell carcinoma- Photomicrograph shows nests of atypical basaloid cells with peripheral palisading. Pigment incontinence seen. [H&E, 20X]



Picture 28: Malignant melanoma- Photomicrograph shows hyperkeratotic epidermis with nests of tumor cells in the dermis. Melanin pigment is seen around the nests. [H&E, 10X]



Picture 29: Malignant melanoma- Photomicrograph shows nests of tumor cells with individual tumor cells showing prominent nucleoli and fine chromation. Melanin pigment is seen within the cells and around the tumor nests. [H&E, 20X]

# KEY TO MASTER CHART

| | |
|---|---|
| DLE | Discoid Lupus erythematosus |
| SLE | Systemic lupus erythematosus |
| DRESS | Drug reaction with eosinophilia and systemic sclerosis |
| BM | Basement membrane |
| NA | Not applicable |
| ECG | Epithelioid cell granuloma |
| BMT | Basement membrane thickening |
| PD | Papillary dermis |
| IHC | Immunohistochemistry |
| ZN | Ziehl- Neelsen |
| VVG | Verhoeff-Van Gieson |
| PAS | Periodic Acid- Schiff |

| S. NO. | NAME | AGE | sex | HISTOPATH NO. | MRD NO. | DOR biopsy | Clinical diagnosis | DURATION | HISTORY OF PRESENT ILLNESS | Mode of onset | Progression | relapse of disease | Aggravating Factors | Morphology of lesion | Patches | Plaques | Papules | Macules | Nodules | Ulcers | Reticular | Vesicles |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | SK | 42 | F | 22-11940 | 1397282 | 14-10-2022 | verruca | 3.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 2 | MK | 36 | F | 22-11893 | 1279959 | 13-10-2022 | warts | 6.0 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 3 | RK | 72 | F | 22-12687 | 1400132 | 04-11-2022 | Lichen Planus/Prurigo nodularis | 20.0 | | Gradual | Present | Absent | Absent | Papules,Nodules | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent |
| 4 | SW | 57 | F | 22-12729 | 22606 | 05-11-2022 | Basal cell carcinoma | 25.0 | | Gradual | Present | Absent | Absent | Nodules | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent |
| 5 | GK | 35 | F | 22-11177 | 1376487 | 24-09-2022 | Warts | 1.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 6 | SH | 47 | M | 22-11127 | 1384012 | 23-09-2022 | verruca | 5.0 | | Gradual | Present | Absent | Absent | Papules,Nodules | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent |
| 7 | BSG | 40 | M | 22-11780 | 1394666 | 10-10-2022 | lichen planus pigmentosus | 2.0 | | Gradual | Present | Absent | Absent | Macules | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 8 | SK | 73 | F | 22-11820 | 1395553 | 11-10-2022 | DLE/sarcoidosis/panniculitis/pseudolymphoma/sweet's syndrome | 0.4 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 9 | JK | 36 | F | 22-12590 | 1407039 | 02-11-2022 | Morphea | 0.7 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 10 | LT | 14 | M | 22-12588 | 216902 | 02-11-2022 | Becker's nevus/morphea | 14.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 11 | SK | 54 | F | 22-12688 | 1199306 | 04-11-2022 | Actinic keratosis | 0.5 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 12 | MA | 44 | F | 22-12077 | 1399487 | 18-10-2022 | Pyoderma gangrenosum | 5.0 | | Gradual | Present | Absent | Absent | Ulcers | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent |
| 13 | SS | 65 | F | 22-10963 | 1381457 | 20-09-2022 | Basal cell carcinoma | 0.5 | | Gradual | Present | Absent | Absent | Ulcers | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent |
| 14 | SK | 68 | M | 22-10965 | 1379144 | 20-09-2022 | Basal cell carcinoma | 1.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 15 | BK | 68 | M | 22-12027 | 582040 | 17-10-2022 | Basal cell carcinoma | 20.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 16 | RK | 29 | F | 22-12692 | 1217872 | 04-11-2022 | lichen planus | 0.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 17 | DK | 54 | M | 22-13082 | 1414179 | 14-11-2022 | lichen planus | 0.7 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 18 | DS | 51 | M | 22- 13322 | 690522 | 19-11-2022 | lichenoid drug rash/psoriasis/erythroderma/drug induced pellagra | 0.1 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 19 | HS | 21 | M | 22-13803 | 1424066 | 01-12-2022 | melanocytic nevus/atypical becker's nevus | 6.0 | | Gradual | Present | Absent | Absent | Macules,Papules | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent |
| 20 | SK | 60 | F | 22-13914 | 1426530 | 05-12-2022 | chronic lichen planus | 6.0 | | Gradual | Present | Absent | Absent | Papules,Nodules | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent |
| 21 | GK | 39 | F | 22-12772 | 1409900 | 07-11-2022 | sarcoidosis/mucinosis | 6.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 22 | RD | 40 | F | 22-12066 | 1399232 | 18-10-2022 | DRESS/ lichenoid drug rash | 0.5 | | Gradual | Present | Absent | Present | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 23 | RD | 40 | F | 22-13804 | 1399232 | 01-Dec | lichen planus/DLE/SLE/Lichenoid polymorphic drug eruption | 0.2 | | Gradual | Present | Present | Present | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 24 | PD | 38 | M | 22-12769 | 1410091 | 07-11-2022 | lichen planus/ lichen planopilaris | 7.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 25 | PD | 38 | M | 22-12770 | 1410091 | 07-11-2022 | lichen planus/ lichen planopilaris | 7.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 26 | SK | 36 | F | 22-12847 | 1411241 | 09-11-2022 | psoriasis lichen planus overlap | 0.2 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 27 | RS | 71 | F | 22-13050 | 1414187 | 14-11-2022 | lichen planus | 0.7 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 28 | SS | 48 | M | 22- 13775 | 1423559 | 30-11-2022 | lichen planus hypertrophicus | 30.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 29 | SR | 40 | F | 22-13279 | 1416782 | 18-11-2022 | Macular amyloidosis | 0.2 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 30 | SK | 69 | F | 22-14058 | 1428417 | 08-12-2022 | Seborrhoeic dermatitis | 0.3 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 31 | BK | 43 | F | 22-14094 | 1428595 | 09-12-2022 | lichen planus/ dermatitis | 9.0 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 32 | MK | 22 | F | 22-14585 | 1435306 | 21-12-2022 | Dermatitis/ Bowenoid papulosis | 3.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 33 | NK | 42 | M | 23-382 | 1444831 | 12-01-2023 | lichen planus/ lichen amyloidosis | 1.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |

| S. NO. | No. of Lesion | Symmetry | Site of lesions | Extremities | Limbs | Trunk/Back | Face area | Scalp | Penis | Size | Distribution of lesion | Extensors | Flexors | Trunk | Face | Generalised | Scalp | Colour | Site of biopsy | HISTOPATHOLOGICAL EXAMINATION OF EPIDERMIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Single | unilateral | right digit of hand | Present | Absent | Absent | Absent | Absent | Absent | 5-6 cm | Right digit of hand | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right hand | |
| 2 | Multiple | Bilateral | feet, wrists | Present | Absent | Absent | Absent | Absent | Absent | 3-4 cm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left foot | |
| 3 | Multiple | Bilateral | Hands, feet, buttocks | Present | Present | Absent | Absent | Absent | Absent | 3-4 mm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | Left hand | |
| 4 | Single | unilateral | left side of nose | Absent | Absent | Absent | Present | Absent | Absent | 5-10 mm | left side of nose | Absent | Absent | Absent | Present | Absent | Absent | blackish | Left side of nose | |
| 5 | Multiple | Bilateral | Legs | Present | Absent | Absent | Absent | Absent | Absent | 6-7 cm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right foot | |
| 6 | Multiple | Bilateral | Arms | Present | Absent | Absent | Absent | Absent | Absent | 2-3 cm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left hand | |
| 7 | Multiple | unilateral | Left axilla,chin | Absent | Absent | Absent | Present | Absent | Absent | 2-3 cm | Left axilla, chin | Absent | Absent | Absent | Present | Absent | Absent | Brownish | chin | |
| 8 | Multiple | Generalised | Forehead | Absent | Absent | Absent | Present | Absent | Absent | 3-4 cm | forehead, face, neck | Absent | Absent | Absent | Present | Absent | Absent | erythematous | Forehead | |
| 9 | Multiple | unilateral | Left Arm | Absent | Present | Absent | Absent | Absent | Absent | 2-3 cm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | violaceous | Left arm | |
| 10 | Single | unilateral | Upper back | Absent | Absent | Present | Absent | Absent | Absent | 8-10 cm | back | Absent | Absent | Present | Absent | Absent | Absent | Brownish | Upper back | |
| 11 | Single | unilateral | left side of nose | Absent | Absent | Absent | Present | Absent | Absent | 4-5 mm | Left side of nose | Absent | Absent | Absent | Present | Absent | Absent | Brownish | Left side of nose | |
| 12 | Multiple | Bilateral | legs, Arms | Absent | Present | Absent | Absent | Absent | Absent | 3-4 cm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | left leg | |
| 13 | Single | unilateral | Left side of forehead | Absent | Absent | Absent | Present | Absent | Absent | 3-4 cm | forehead | Absent | Absent | Absent | Present | Absent | Absent | Brownish | left side of forehead | |
| 14 | Single | unilateral | Forehead | Absent | Absent | Absent | Present | Absent | Absent | 2-3 cm | Forehead | Absent | Absent | Absent | Present | Absent | Absent | blackish | forehead | |
| 15 | Single | unilateral | Upper back | Absent | Absent | Present | Absent | Absent | Absent | 3-4 cm | Upper back | Absent | Absent | Present | Absent | Absent | Absent | Brownish | back | |
| 16 | Multiple | Bilateral | Thigh | Absent | Present | Absent | Absent | Absent | Absent | 3 cm | Flexors | Absent | Present | Absent | Absent | Absent | Absent | Brownish | left thigh | |
| 17 | Multiple | Bilateral | extremities, Face, Neck | Present | Absent | Absent | Present | Absent | Absent | 2-3mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | left arm | |
| 18 | Multiple | Bilateral | all over the body | Present | Absent | Absent | Absent | Absent | Absent | 3-4 mm | entire body | Absent | Absent | Absent | Absent | Present | Absent | Brownish | left arm | |
| 19 | Multiple | unilateral | blaschko line pattern | Present | Absent | Absent | Absent | Absent | Absent | 3-4 mm | right calf | Present | Absent | Absent | Absent | Absent | Absent | blackish | right calf | |
| 20 | Multiple | Bilateral | extensors | Present | Absent | Absent | Absent | Absent | Absent | 4-5 mm | extremities, trunk | Present | Absent | Present | Absent | Absent | Absent | violaceous | back | |
| 21 | Multiple | Generalised | back, Neck, shoulders | Absent | Absent | Present | Absent | Absent | Absent | 3-5 mm | back, neck, shoulders | Absent | Absent | Present | Absent | Absent | Absent | Brownish | back | |
| 22 | Multiple | Bilateral | extremities, Trunk, Face | Present | Absent | Present | Present | Absent | Absent | 4-5 cm | Extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right leg | |
| 23 | Multiple | Bilateral | extremities, Face, Neck | Present | Absent | Absent | Present | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right arm | |
| 24 | Multiple | Bilateral | extremities, scalp, abdomen | Present | Absent | Absent | Absent | Present | Absent | variable | extensors, flexors | Present | Present | Absent | Absent | Absent | Absent | Brownish | scalp | |
| 25 | Multiple | Bilateral | extremities, scalp, abdomen | Present | Absent | Absent | Absent | Present | Absent | variable | extensors, flexors | Present | Present | Absent | Absent | Absent | Absent | Brownish | abdomen | |
| 26 | Multiple | Generalised | abdomen | Absent | Absent | Present | Absent | Absent | Absent | 4-5 cm | abdomen | Absent | Absent | Present | Absent | Absent | Absent | erythematous | abdomen | |
| 27 | Multiple | Bilateral | Hands, feet, buttocks | Absent | Present | Absent | Absent | Absent | Absent | 3-4 mm | extensors, flexors | Present | Present | Absent | Absent | Absent | Absent | erythematous | right hand | |
| 28 | Multiple | Bilateral | extremities | Present | Absent | Absent | Absent | Absent | Absent | 2-3 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | left leg | |
| 29 | Multiple | Bilateral | extremities, Trunk | Present | Absent | Present | Absent | Absent | Absent | 2-4 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right leg | |
| 30 | Single | unilateral | Thigh | Absent | Present | Absent | Absent | Absent | Absent | 4-5 mm | Flexors | Absent | Present | Absent | Absent | Absent | Absent | blackish | right thigh | |
| 31 | Multiple | Bilateral | extremities, scalp, forehead | Present | Absent | Absent | Absent | Present | Absent | 3-4 mm | extremities, scalp, forehead | Present | Absent | Absent | Absent | Absent | Present | violaceous | scalp | |
| 32 | Multiple | Generalised | penile shaft | Absent | Absent | Absent | Absent | Absent | Present | 5-6 mm | penile shaft | Absent | Absent | Absent | Absent | Present | Absent | Brownish | penis | |
| 33 | Multiple | Bilateral | extremities,abdomen, buttocks | Present | Absent | Absent | Absent | Absent | Absent | 1-2 mm | extensors, flexors | Present | Present | Absent | Absent | Absent | Absent | Brownish | right leg | |

| S. NO. | Hyperkeratosis | Parakeratosis | Hypergranulosis | Papillomatosis | Acanthosis | Thinning | Spongiosis | Basal cell degeneration | Civatte Bodies | Max Joseph Space | Saw Toothed Rete Ridges | Flattening of rete ridges | Inflammatory infiltrate | Follicular Plugging | Thickening of BM | Pigment in Basal Layer | Horn Cyst | Squamous Eddies | Necrotic Keratocytes | Ulceration | Dysplasia |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 2 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 3 | Present | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 4 | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 5 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 6 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 7 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 8 | Present | Present | Absent | Absent | Present | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 9 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 10 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 11 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 12 | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 13 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 14 | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 15 | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 16 | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 17 | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 18 | Absent | Absent | Absent | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 19 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 20 | Present | Absent | Absent | Absent | Present | Absent | Absent | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 21 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 22 | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 23 | Present | Present | Present | Absent | Present | Absent | Absent | Present | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 24 | Present | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 25 | Present | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 26 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 27 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 28 | Present | Absent | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 29 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 30 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 31 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 32 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 33 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |

| S. NO. | Malignant cells | HISTOPATHOLOGICAL EXAMINATION OF DERMIS | INFILTRATION | Location | Type of Cells | Band like Inflammatory Infilterate | Perivascular infiltrate | Periadenexal Infiltrate | ECG | Pigment Incontinence | BMT | Degenerated collagen | Inc Collagenisation | Fibrosis in PD | Amyloid Deposits in PD | NEVUS CELLS | NEVUS CELLS LOCATION |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 2 | Absent | | Present | Superficial dermis | lymphocytes,epithelioid cells, histiocytic collections, giant cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 3 | Absent | | Present | Deep dermis | lymphocytes, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 4 | Absent | | Present | Superficial dermis | Tumor cells | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 5 | Absent | | Present | Deep dermis | lymphocytes,plasma cells,eosinophils,histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 6 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 7 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 8 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 9 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | NA |
| 10 | Absent | | Present | Superficial dermis | melanophages | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 11 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 12 | Absent | | Present | Superficial dermis | lymphocytes,plasma cells,neutrophils,histiocytes | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 13 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 14 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 15 | Absent | | Present | Superficial dermis | Tumor cells | Absent | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 16 | Absent | | Present | Superficial dermis | Tumor cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 17 | Absent | | Present | Superficial dermis | Tumor cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 18 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Present | Dermis |
| 19 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells, melanophages | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Dermis |
| 20 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Dermis |
| 21 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Dermis |
| 22 | Absent | | Present | Deep dermis | lymphocytes, eosinophils, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 23 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 24 | Absent | | Present | Deep dermis | lymphocytes | Present | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 25 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 26 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 27 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 28 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 29 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | NA |
| 30 | Absent | | Absent | Superficial dermis | Lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 31 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Present | Absent | Absent | NA |
| 32 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | NA |
| 33 | Absent | | Present | Superficial dermis | lymohocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |

| S. NO. | NEVUS CELLS MATURATION | NEVUS CELLS NECROSIS | NEVUS CELLS MITOSIS | NEVUS CELLS JUNCTIONAL ACTIVITY | MELANOMA CELLS | MELANOMA CELLS NECROSIS | MELANOMA CELLSVERTICAL SPREAD | MELANOMA CELLS HORIZONTAL SPREAD | ANY OTHER MALIGNANT CELLS | SUBCUTANEOUS TISSUE | INFLAMMATORY INFILTRATE | TYPE OF CELLS | SPECIAL STAINS | IHC | FINAL DIAGNOSIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | congo red- apple green birefringence | NA | Amyloidosis |
| 2 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Present | Absent | NA | NA | NA | NA | Basal cell carcinoma |
| 3 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Present | Absent | NA | NA | NA | NA | Basal cell carcinoma |
| 4 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Present | Absent | NA | NA | NA | NA | Basal cell carcinoma |
| 5 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Present | Absent | NA | NA | NA | NA | Basal cell carcinoma |
| 6 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | Absent | NA | NA | NA | Becker's nevus |
| 7 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Becker's nevus |
| 8 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Present | Absent | NA | NA | NA | Becker's nevus |
| 9 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid lupus erythematosus |
| 10 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid lupus erythematosus |
| 11 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid lupus erythematosus |
| 12 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid lupus erythematosus |
| 13 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid lupus erythematosus |
| 14 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid lupus erythematosus |
| 15 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | 5% ZN, 20% Zn, reticulin- negative | NA | Discoid lupus erythematosus |
| 16 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Discoid Lupus erythematosus |
| 17 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Fixed drug eruption |
| 18 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Intradermal nevus |
| 19 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Intradermal nevus |
| 20 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Intradermal nevus |
| 21 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Intradermal nevus |
| 22 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planopilaris |
| 23 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planopilaris |
| 24 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planopilaris |
| 25 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen Planopilaris |
| 26 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planopilaris |
| 27 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planopilaris |
| 28 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planopilaris |
| 29 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | lichen planopilaris |
| 30 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | lichen planopilaris |
| 31 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 32 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 33 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |

| S. NO. | NAME | AGE | sex | HISTOPATH NO. | MRD NO. | DOR biopsy | Clinical diagnosis | DURATION | HISTORY OF PRESENT ILLNESS | Mode of onset | Progression | relapse of disease | Aggravating Factors | Morphology of lesion | Patches | Plaques | Papules | Macules | Nodules | Ulcers | Reticular | Vesicles |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | HS | 33 | M | 23-1371 | 1443548 | 10-02-2023 | tinea capitis/verrucous epidermal nevus | 4.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 35 | VC | 45 | F | 23-1590 | 1463780 | 15-02-2023 | Granuloma annulare | 1.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 36 | SK | 58 | F | 23-1634 | 1023916 | 16-02-2023 | lichen planus hypertrophicus/psoriasis vulgaris | 2.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 37 | DS | 51 | M | 23-1714 | 1452696 | 18-02-2023 | systemic sclerosis | 0.4 | | Gradual | Present | Present | Absent | Macules | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 38 | RD | 38 | F | 23-1766 | 1466250 | 20-02-2023 | palmoplantar eczema/ palmoplantar psoriasis | 1.0 | | Gradual | Present | Present | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 39 | PK | 47 | M | 23-2162 | 1017365 | 01-03-2023 | DLE | 6.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 40 | SS | 67 | M | 23-2164 | 245199 | 01-03-2023 | lichen planus hypertrophicus | 0.3 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 41 | KD | 30 | F | 23-2347 | 1474660 | 06-03-2023 | Tubercular tuberculoid leprosy | 5.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 42 | KK | 50 | F | 23-2767 | 1463608 | 15-03-2023 | lichen planus pigmentosus/ fixed drug eruption | 1.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 43 | JS | 83 | M | 23-2775 | 1427148 | 15-03-2023 | seborrhoeic keratosis | 10.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 44 | SS | 57 | F | 23-2883 | 1460654 | 17-03-2023 | lichen planus pigmentosus/macular amyloidosis | 2.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 45 | SR | 34 | F | 23-3407 | 1488107 | 24-03-2023 | lichen planus pigmentosus | 7.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 46 | JK | 23 | F | 23-3665 | 1492188 | 04-04-2023 | Disseminated DLE | 0.2 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 47 | NS | 5 | F | 23-3727 | 1481869 | 05-04-2023 | Lichen Planus with extensive PIH/ Atopic dermatitis | 0.3 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 48 | SS | 41 | M | 23-3762 | 1125680 | 06-04-2023 | Lichen Planus | 0.1 | | Gradual | Present | Absent | Absent | Macules | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 49 | SR | 38 | F | 23-4008 | 1496341 | 12-04-2023 | Post burn dysplasia/ Squamous cell carcinoma | 0.5 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 50 | AS | 36 | M | 23-4172 | 1500185 | 17-04-2023 | lichen planus pigmentosus/ pigmentary contact dermatitis | 0.5 | | Gradual | Absent | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 51 | AK | 21 | F | 23-4228 | 1501134 | 18-04-2023 | Pityriasis lichenoides chronica | 4.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 52 | GK | 43 | F | 23-4433 | 1269204 | 21-04-2023 | Livedo reticularis | 2.0 | | Gradual | Present | Absent | Absent | Plaques,reticular | Absent | Present | Absent | Absent | Absent | Absent | Present | Absent |
| 53 | AS | 23 | F | 23-4435 | 1501811 | 21-04-2023 | lichen planus pigmentosus/ PIH | 1.0 | | Gradual | Present | Absent | Absent | Macules | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 54 | CK | 40 | M | 23-4530 | 1504315 | 24-04-2023 | hyperkeratotic eczema/ palmoplantar psoriasis | 4.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 55 | RC | 62 | M | 23-4695 | 1506419 | 27-04-2023 | seborrhoeic dermatitis | 0.5 | | Gradual | Absent | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 56 | SK | 55 | F | 23-4702 | 1506442 | 27-04-2023 | lichen planus pigmentosus | 6.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 57 | RA | 65 | F | 23-5348 | 1515139 | 11-05-2023 | lichen planus | 0.0 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 58 | VA | 75 | M | 23-5451 | 362980 | 13-05-2023 | endogenous eczema | 20.0 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 59 | DK | 58 | F | 23-6104 | 1524544 | 27-05-2023 | lichen planus pigmentosus | 4.0 | | Gradual | Present | Absent | Absent | Macules,Patches | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 60 | RJ | 29 | F | 23-6235 | 1527083 | 31-05-2023 | prurigo nodularis/ pre bullous stage of bullous pemphigoid | 2.0 | | Gradual | Present | Absent | Absent | Papules,Nodules | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent |
| 61 | SG | 58 | M | 23-6327 | 1528368 | 02-06-2023 | seborrhoeic dermatosis/ Solar lentigo | 4.0 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 62 | MK | 3 | F | 23-6736 | 1530776 | 10-06-2023 | Linear scleroderma/ Morphea | 0.3 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 63 | NR | 30 | F | 23-7284 | 1539483 | 21-06-2023 | Lichen planopilaris | 0.5 | | Gradual | Present | Absent | Absent | Macules | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 64 | HK | 48 | F | 23-7286 | 1539787 | 21-06-2023 | Livedo reticularis | 0.5 | | Gradual | Present | Absent | Absent | Plaques,reticular | Absent | Present | Absent | Absent | Absent | Absent | Present | Absent |
| 65 | KT | 30 | M | 23-9150 | 1563525 | 02-08-2023 | Secondary syphilis/ Lichen planus- DLE overlap | 0.2 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 66 | SK | 49 | F | 23-9706 | 1570715 | 14-08-2023 | Eruptive Lichen planus/ Lichenoid eruption | 2.0 | | Gradual | Present | Absent | Present | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |

| S. NO. | No. of Lesion | Symmetry | Site of lesions | Extremities | Limbs | Trunk/Back | Face area | Scalp | Penis | Size | Distribution of lesion | Extensors | Flexors | Trunk | Face | Generalised | Scalp | Colour | Site of biopsy | HISTOPATHOLOGICAL EXAMINATION OF EPIDERMIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | Single | unilateral | right side of scalp | Absent | Absent | Absent | Absent | Present | Absent | 4-5 cm | scalp | Absent | Absent | Absent | Absent | Absent | Present | erythematous | right side of scalp | |
| 35 | Multiple | Bilateral | dorsum of hands, forehead, back | Absent | Present | Present | Present | Absent | Absent | 3-4 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | dorsum of left hand | |
| 36 | Multiple | Bilateral | bilateral medial malleolus | Present | Absent | Absent | Absent | Absent | Absent | 5-6 mm | ankle | Present | Absent | Absent | Absent | Absent | Absent | violaceous | left medial malleolus of ankle | |
| 37 | Multiple | Bilateral | extremities, abdomen | Present | Absent | Absent | Absent | Absent | Absent | 5-6 mm | extensors, flexors | Present | Present | Absent | Absent | Absent | Absent | Brownish | abdomen | |
| 38 | Single | Bilateral | palmar as[ect of both hands | Absent | Present | Absent | Absent | Absent | Absent | 7-8 cm | hands | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right hand | |
| 39 | Multiple | Bilateral | nose, ears, scalp | Absent | Absent | Absent | Present | Present | Absent | 2-4 cm | nose, ears, scalp | Absent | Absent | Absent | Present | Absent | Absent | Brownish | scalp | |
| 40 | Single | unilateral | Legs | Absent | present | Absent | Absent | Absent | Absent | 1 cm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right leg | |
| 41 | Single | unilateral | elbow | Absent | present | Absent | Absent | Absent | Absent | 3-6 cm | Flexors | Absent | Present | Absent | Absent | Absent | Absent | Brownish | right elbow | |
| 42 | Multiple | Bilateral | feet, wrists | Absent | Present | Absent | Absent | Absent | Absent | 3-4 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right foot | |
| 43 | Multiple | Generalised | Face, abdomen | Absent | Absent | Absent | Present | Absent | Absent | 5-6 mm | face, abdomen | Absent | Absent | Absent | Present | Absent | Absent | blackish | face (left cheek) | |
| 44 | Multiple | Generalised | back, Neck | Absent | Absent | Present | Absent | Absent | Absent | 4-5 mm | back, neck, shoulders | Absent | Absent | Present | Absent | Absent | Absent | blackish | back | |
| 45 | Multiple | Bilateral | Face, Arms, legs | Absent | Present | Absent | Present | Absent | Absent | 3-4 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right arm | |
| 46 | Multiple | Bilateral | Face, hands, feet | Absent | Present | Absent | Present | Absent | Absent | 1-2 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | left foot | |
| 47 | Multiple | Bilateral | knee, legs, back, Trunk | Absent | Absent | Present | Absent | Absent | Absent | 3-4 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left knee | |
| 48 | Multiple | Bilateral | foot, back, Trunk, buttocks | Absent | Absent | Present | Absent | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | violaceous | left foot | |
| 49 | Single | unilateral | Thigh | Absent | Present | Absent | Absent | Absent | Absent | 4-5 cm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right thigh | |
| 50 | Multiple | Bilateral | cheeks, forehead, Neck | Absent | Absent | Absent | Present | Absent | Absent | 5-6 mm | cheeks, forehead, neck | Absent | Absent | Absent | Present | Absent | Absent | violaceous | left cheek | |
| 51 | Multiple | Bilateral | Arms, Trunk | Absent | Present | Present | Absent | Absent | Absent | 3-4 mm | flexors | Absent | Present | Absent | Absent | Absent | Absent | erythematous | left arm | |
| 52 | Multiple | Bilateral | lower limbs | Absent | Present | Absent | Absent | Absent | Absent | 6-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | right leg | |
| 53 | Multiple | Bilateral | Arms, wrists | Present | Absent | Absent | Absent | Absent | Absent | 4-5 mm | flexors | Absent | Present | Absent | Absent | Absent | Absent | blackish | left hand | |
| 54 | Multiple | Bilateral | palms of both hands | Present | Absent | Absent | Absent | Absent | Absent | 3- 6 mm | flexors | Absent | Present | Absent | Absent | Absent | Absent | blackish | left palm | |
| 55 | Single | unilateral | frontal aspect of scalp | Absent | Absent | Absent | Absent | Present | Absent | 5 cm | scalp | Absent | Absent | Absent | Absent | Absent | Present | blackish | Frontal aspect of scalp | |
| 56 | Single | Generalised | Neck, Face | Absent | Absent | Absent | Present | Absent | Absent | 6-7 cm | neck, face | Absent | Absent | Absent | Present | Absent | Absent | blackish | back of neck | |
| 57 | Multiple | Generalised | back | Absent | Absent | Present | Absent | Absent | Absent | 5-6 mm | back | Absent | Absent | Present | Absent | Absent | Absent | blackish | back | |
| 58 | Multiple | Bilateral | Arms, legs | Absent | Present | Absent | Absent | Absent | Absent | 5-10 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left arm | |
| 59 | Multiple | Bilateral | Face, Arms | Absent | Present | Absent | Present | Absent | Absent | 5-6 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | face | |
| 60 | Multiple | Bilateral | Arms, legs, Trunk | Absent | Present | Present | Absent | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | left leg | |
| 61 | Multiple | unilateral | scalp, Face | Absent | Absent | Absent | Present | Present | Absent | 5-6 mm | scalp, face | Absent | Absent | Absent | Present | Absent | Present | blackish | scalp | |
| 62 | Single | unilateral | Legs | Absent | Present | Absent | Absent | Absent | Absent | 7-8 cm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | Right leg | |
| 63 | Multiple | Bilateral | forehead, Arms, legs | Absent | Present | Absent | Present | Absent | Absent | 4-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | forehead | |
| 64 | Single | unilateral | Arm | Absent | Present | Absent | Absent | Absent | Absent | 8-10 mm | flexors | Absent | Present | Absent | Absent | Absent | Absent | blackish | right arm | |
| 65 | Multiple | Bilateral | thighs, Arms, abdomen,back | Absent | Present | Present | Absent | Absent | Absent | 5-6 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | right thigh | |
| 66 | Multiple | Bilateral | legs, Arms, lower abdomen | Absent | Present | Present | Absent | Absent | Absent | 7-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left leg | |

| S. NO. | Hyperkeratosis | Parakeratosis | Hypergranulosis | Papillomatosis | Acanthosis | Thinning | Spongiosis | Basal cell degeneration | Civatte Bodies | Max Joseph Space | Saw Toothed Rete Ridges | Flattening of rete ridges | Inflammatory infiltrate | Follicular Plugging | Thickening of BM | Pigment in Basal Layer | Horn Cyst | Squamous Eddies | Necrotic Keratocytes | Ulceration | Dysplasia |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 35 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 36 | Present | Absent | Absent | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 37 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 38 | Present | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 39 | Present | Absent | Absent | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 40 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent |
| 41 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 42 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 43 | Present | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent |
| 44 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 45 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 46 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 47 | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 48 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 49 | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 50 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 51 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 52 | Present | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 53 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 54 | Present | Present | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 55 | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent |
| 56 | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 57 | Present | Present | Present | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 58 | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 59 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 60 | Present | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 61 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 62 | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 63 | Present | Absent | Present | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 64 | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 65 | Absent | Absent | Absent | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 66 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |

| S. NO. | Malignant cells | HISTOPATHOLOGICAL EXAMINATION OF DERMIS | INFILTRATION | Location | Type of Cells | Band like Inflammatory Infilterate | Perivascular infiltrate | Periadenexal Infiltrate | ECG | Pigment Incontinence | BMT | Degenerated collagen | Inc Collagenisation | Fibrosis in PD | Amyloid Deposits in PD | NEVUS CELLS | NEVUS CELLS LOCATION |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | Absent | | Not included | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | NA | Absent | NA |
| 35 | Absent | | Present | Deep dermis | lymphocytes, histiocytes, giant cells | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | NA |
| 36 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 37 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | NA |
| 38 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 39 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 40 | Absent | | Absent | NA | NA | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 41 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 42 | Absent | | Absent | NA | NA | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 43 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 44 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 45 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 46 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 47 | Absent | | Present | Deep dermis | lymphocytes, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 48 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 49 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 50 | Absent | | Present | Deep dermis | lymphocytes, histiocytes | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 51 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 52 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 53 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 54 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 55 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 56 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 57 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Present | Present | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 58 | Absent | | Present | Superficial dermis | lymphocytes, neutrophils, histiocytes | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 59 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 60 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | NA |
| 61 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 62 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | NA |
| 63 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 64 | Absent | | Absent | NA | NA | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 65 | Absent | | Absent | NA | NA | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 66 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |

| S. NO. | NEVUS CELLS MATURATION | NEVUS CELLS NECROSIS | NEVUS CELLS MITOSIS | NEVUS CELLS JUNCTIONAL ACTIVITY | MELANOMA CELLS | MELANOMA CELLS NECROSIS | MELANOMA CELLSVERTICAL SPREAD | MELANOMA CELLS HORIZONTAL SPREAD | ANY OTHER MALIGNANT CELLS | SUBCUTANEOUS TISSUE | INFLAMMATORY INFILTRATE | TYPE OF CELLS | SPECIAL STAINS | IHC | FINAL DIAGNOSIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 34 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 35 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 36 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 37 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 38 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 39 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 40 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 41 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 42 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 43 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 44 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen Planus |
| 45 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen planus |
| 46 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | lichen planus hypertrophicus |
| 47 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | lichen planus hypertrophicus |
| 48 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | lichen planus hypertrophicus |
| 49 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | lichen planus hypertrophicus |
| 50 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 51 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 52 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 53 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | 20% ZN- Negative | NA | Lichen simplex chronicus |
| 54 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 55 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 56 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 57 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | PAS- negative | NA | Lichen simplex chronicus |
| 58 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 59 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichen simplex chronicus |
| 60 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Present | Present | lymphocytes, plasma cells, sclerosis | NA | NA | Lichen planus pigmentosus |
| 61 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | 5% ZN- negative | NA | Lichenoid drug eruption |
| 62 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichenoid drug eruption |
| 63 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | Toluidine blue- mast cells | NA | Lichenoid drug eruption |
| 64 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Present | Absent | NA | NA | NA | Lichenoid drug eruption |
| 65 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | 5% ZN- negative | NA | Lichenoid drug eruption |
| 66 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichenoid drug eruption |

| S. NO. | NAME | AGE | sex | HISTOPATH NO. | MRD NO. | DOR biopsy | Clinical diagnosis | DURATION | HISTORY OF PRESENT ILLNESS | Mode of onset | Progression | relapse of disease | Aggravating Factors | Morphology of lesion | Patches | Plaques | Papules | Macules | Nodules | Ulcers | Reticular | Vesicles |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | PC | 31 | M | 23-9888 | 351689 | 18-08-2023 | Verucca vulgaris/Tuberculosis verrucosa cutis | 0.5 | | Gradual | Present | Absent | Present | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 68 | AG | 35 | F | 23-10030 | 1575078 | 21-08-2023 | Lichen Planopilaris | 0.3 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 69 | NK | 43 | F | 23-10072 | 928949 | 22-08-2023 | Lichen planus pigmentosus/ Riehl's melanosis | 3.0 | | Gradual | Present | Absent | Present | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 70 | NK | 28 | M | 23-10074 | 1400525 | 22-08-2023 | Mycosis fungoides | 1.5 | | Gradual | Present | Present | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 71 | JS | 76 | M | 23-10171 | 1540142 | 24-08-2023 | Lupus vulgaris/ Lupus erythematosus/ Cutaneous sarcoidosis | 20.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 72 | RD | 46 | F | 23-10224 | 720386 | 25-08-2023 | lichen planus | 0.8 | | Gradual | Present | Absent | Absent | Bullae,Vesicles | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present |
| 73 | HS | 23 | M | 23-10580 | 1578637 | 02-09-2023 | Prurigo nodularis/ TB gumma/ Lichen planus hypertrophicus | 1.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 74 | SR | 60 | F | 24-43 | 1633532 | 02-01-2024 | Psoariasis/ Lichen amyloidosis | 20.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 75 | NS | 32 | F | 24-133 | 224046 | 04-01-2024 | Lichen Planus/ Plane warts | 6.0 | | Gradual | Present | Absent | Absent | Papules,Plaques | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent |
| 76 | AS | 40 | M | 24-174 | 1648588 | 05-01-2024 | Discoid eczema/ Lichen simplex chronicus | 15.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 77 | SG | 55 | M | 24-273 | 1650097 | 08-01-2024 | lichen planus | 0.5 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 78 | BK | 39 | F | 24-282 | 1650297 | 08-01-2024 | lichen planus | 1.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 79 | RL | 50 | M | 24-433 | 1652646 | 13-01-2024 | Disseminated DLE | 1.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 80 | RJ | 38 | F | 24-589 | 1655019 | 18-01-2024 | Lichen planus pigmentosus | 3.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 81 | DK | 38 | F | 24-805 | 1646741 | 24-01-2024 | Pemphigus vulgaris | 0.2 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 82 | JK | 61 | F | 24-918 | 1616333 | 21-01-2024 | DLE | 5.0 | | Sudden | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 83 | RS | 32 | M | 24-958 | 1660848 | 29-01-2024 | Endogenous eczema/ Dermatitis herpetiformis | 4.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 84 | JK | 12 | F | 24-962 | 1661018 | 29-01-2024 | Morphea | 0.2 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 85 | AK | 58 | F | 24-1073 | 1443008 | 31-01-2024 | Sclerosing Panniculitis | 1.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 86 | DK | 24 | M | 24-1190 | 1664291 | 03-02-2024 | Pityriasis rubra pilaris/ Tinea corporis | 10.0 | | Gradual | Present | Absent | Absent | Patches | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 87 | SS | 65 | M | 24-2372 | 1684297 | 01-03-2024 | Prurigo nodularis | 0.3 | | Gradual | Present | Absent | Absent | Nodules | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent |
| 88 | MA | 51 | F | 24-2484 | 1685856 | 04-03-2024 | Papilloma | 8.0 | | Gradual | Present | Absent | Absent | Nodules | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent |
| 89 | MS | 57 | M | 24-2624 | 1544369 | 07-03-2024 | BCC/ Malignant neoplasm | 6.0 | | Gradual | Present | Absent | Absent | Papules | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent |
| 90 | DK | 71 | M | 24-2774 | 364919 | 11-03-2024 | melanoma | 5.0 | | Gradual | Present | Absent | Absent | Plaques | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent |

| S. NO. | No. of Lesion | Symmetry | Site of lesions | Extremities | Limbs | Trunk/Back | Face area | Scalp | Penis | Size | Distribution of lesion | Extensors | Flexors | Trunk | Face | Generalised | Scalp | Colour | Site of biopsy | HISTOPATHOLOGICAL EXAMINATION OF EPIDERMIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | Single | unilateral | little toe | Present | Absent | Absent | Absent | Absent | Absent | 4-5 cm | toe | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left little toe | |
| 68 | Single | unilateral | Neck, temporal region | Absent | Absent | Absent | Present | Absent | Absent | 10 cm | right temporal region | Absent | Absent | Absent | Absent | Absent | Present | blackish | scalp | |
| 69 | Multiple | Generalised | Face, Neck | Absent | Absent | Absent | Present | Absent | Absent | 10-15 cm | face, neck | Absent | Absent | Absent | Present | Absent | Absent | Brownish | scalp | |
| 70 | Multiple | Bilateral | back, Arms | Absent | Present | Present | Absent | Absent | Absent | 8-10 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | right arm | |
| 71 | Single | unilateral | buttock | Absent | Absent | Present | Absent | Absent | Absent | 5-7 cm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right buttock | |
| 72 | Multiple | Bilateral | Arms, legs, back | Absent | Present | Present | Absent | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | right thigh | |
| 73 | Multiple | Bilateral | Face, body | Absent | Absent | Absent | Present | Absent | Absent | 1-2 cm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | right thigh | |
| 74 | Multiple | Bilateral | lower limbs | Absent | Present | Absent | Absent | Absent | Absent | 6-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | left lower limb | |
| 75 | Multiple | unilateral | Face, right Arm | Absent | Present | Absent | Present | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right arm | |
| 76 | Single | unilateral | right leg | Absent | Present | Absent | Absent | Absent | Absent | 7-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right leg | |
| 77 | Multiple | Generalised | body | Present | Absent | Absent | Absent | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | chest | |
| 78 | Multiple | Generalised | body | Present | Absent | Absent | Absent | Absent | Absent | 6-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right leg | |
| 79 | Multiple | Bilateral | Face, ears, chest, Arms | Absent | Present | Absent | Present | Absent | Absent | 8-10 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | chest | |
| 80 | Single | unilateral | left lateral side of nose | Absent | Absent | Absent | Present | Absent | Absent | 6-7 mm | nose | Absent | Absent | Absent | Present | Absent | Absent | Brownish | Left lateral side of nose | |
| 81 | Multiple | Bilateral | legs, abdomen, back | Absent | Absent | Present | Absent | Absent | Absent | 8-10 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | right leg | |
| 82 | Single | unilateral | occipital area of scalp | Absent | Absent | Absent | Absent | Present | Absent | 2-3 cm | occipital area of scalp | Absent | Absent | Absent | Absent | Absent | Present | violaceous | Scalp | |
| 83 | Multiple | Bilateral | Arms, legs, back, abdomen | Absent | Present | Present | Absent | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | violaceous | left arm | |
| 84 | Single | Generalised | Lateral aspect of thigh | Absent | Present | Absent | Absent | Absent | Absent | 5-6 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | Left thigh | |
| 85 | Multiple | Bilateral | Legs | Absent | Present | Absent | Absent | Absent | Absent | 9-10 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left leg | |
| 86 | Multiple | Bilateral | knees, feet, groin folds | Absent | Present | Absent | Absent | Absent | Absent | 6-8 mm | flexors | Absent | Present | Absent | Absent | Absent | Absent | Brownish | right knee | |
| 87 | Multiple | Bilateral | back, legs, thighs, Arms, buttocks | Absent | Present | Present | Absent | Absent | Absent | 6-8 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | erythematous | back | |
| 88 | Single | unilateral | Thigh | Absent | Present | Absent | Absent | Absent | Absent | 1-2 cm | extensors | Present | Absent | Absent | Absent | Absent | Absent | Brownish | left thigh | |
| 89 | Multiple | Bilateral | Legs | Present | Present | Absent | Absent | Absent | Absent | 4-5 mm | extensors | Present | Absent | Absent | Absent | Absent | Absent | blackish | left leg | |
| 90 | Multiple | Unilateral | Right foot | Present | Absent | Present | Absent | Absent | Absent | 4-5 mm | Extensors | Present | Absent | Present | Absent | Absent | Absent | Brownish | right foot | |

| S. NO. | Hyperkeratosis | Parakeratosis | Hypergranulosis | Papillomatosis | Acanthosis | Thinning | Spongiosis | Basal cell degeneration | Civatte Bodies | Max Joseph Space | Saw Toothed Rete Ridges | Flattening of rete ridges | Inflammatory infiltrate | Follicular Plugging | Thickening of BM | Pigment in Basal Layer | Horn Cyst | Squamous Eddies | Necrotic Keratocytes | Ulceration | Dysplasia |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 68 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 69 | Present | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 70 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 71 | Present | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 72 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 73 | Present | Present | Present | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 74 | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 75 | Absent | Absent | Absent | Absent | Present | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 76 | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 77 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 78 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 79 | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent |
| 80 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 81 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 82 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 83 | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 84 | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 85 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent |
| 86 | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 87 | Present | Present | Absent | Absent | Present | Absent | Present | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 88 | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 89 | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |
| 90 | Present | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent |

| S. NO. | Malignant cells | HISTOPATHOLOGICAL EXAMINATION OF DERMIS | INFILTRATION | Location | Type of Cells | Band like Inflammatory Infilterate | Perivascular infiltrate | Periadenexal Infiltrate | ECG | Pigment Incontinence | BMT | Degenerated collagen | Inc Collagenisation | Fibrosis in PD | Amyloid Deposits in PD | NEVUS CELLS | NEVUS CELLS LOCATION |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 68 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 69 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 70 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 71 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells, histiocytes | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 72 | Absent | | Present | Superficial dermis | lymphocytes, neutrophils, eosinophils | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 73 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 74 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 75 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells, neutrophils | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 76 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 77 | Absent | | Present | Superficial dermis | lymphgocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 78 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 79 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 80 | Absent | | Absent | NA | NA | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 81 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 82 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | NA |
| 83 | Absent | | Present | Superficial and deep dermis | lymphocytes, plasma cells, neutrophils, histiocytes | Absent | Present | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 84 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Present | Absent | Absent | Absent | NA |
| 85 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 86 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 87 | Absent | | Present | Deep dermis | lymphocytes, plasma cells, eosinophils, histiocytes | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 88 | Absent | | Present | Deep dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | Absent | NA |
| 89 | Absent | | Present | Deep dermis | lymohocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | NA |
| 90 | Absent | | Present | Superficial dermis | lymphocytes, plasma cells | Absent | Present | Absent | Absent | Present | Absent | Absent | Absent | Absent | Absent | Absent | NA |

| S. NO. | NEVUS CELLS MATURATION | NEVUS CELLS NECROSIS | NEVUS CELLS MITOSIS | NEVUS CELLS JUNCTIONAL ACTIVITY | MELANOMA CELLS | MELANOMA CELLS NECROSIS | MELANOMA CELLSVERTICAL SPREAD | MELANOMA CELLS HORIZONTAL SPREAD | ANY OTHER MALIGNANT CELLS | SUBCUTANEOUS TISSUE | INFLAMMATORY INFILTRATE | TYPE OF CELLS | SPECIAL STAINS | IHC | FINAL DIAGNOSIS |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 67 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Lichenoid drug eruption |
| 68 | NA | Absent | Absent | Absent | present | Absent | absent | present | Absent | Absent | NA | NA | NA | NA | Malignant melanoma |
| 69 | NA | Absent | Absent | Absent | present | Absent | absent | present | Absent | Absent | NA | NA | NA | NA | Malignant melanoma |
| 70 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | Absent | NA | NA | NA | Morphea |
| 71 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Morphea |
| 72 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | VVG- Positive | NA | Morphea |
| 73 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Postinflammatory hyperpigmentation |
| 74 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Postinflammatory hyperpigmentation |
| 75 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Postinflammatory hyperpigmentation |
| 76 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Postinflammatory hyperpigmentation |
| 77 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Postinflammatory hyperpigmentation |
| 78 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Prurigo nodularis |
| 79 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Prurigo nodularis |
| 80 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | seborrhoeic keratosis |
| 81 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | seborrhoeic keratosis |
| 82 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | seborrhoeic keratosis |
| 83 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | seborrhoeic keratosis |
| 84 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Subacute spongiotic dermatitis |
| 85 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Subacute spongiotic dermatitis |
| 86 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | PAS- negative | NA | Verruca vulgaris |
| 87 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Verruca vulgaris |
| 88 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Verruca vulgaris |
| 89 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | NA | NA | Verruca vulgaris |
| 90 | NA | Absent | Absent | Absent | Absent | Absent | NA | NA | Absent | Absent | NA | NA | PAS- negative | NA | Verrucous epidermal nevus |

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No. BFUHS/2K23p-TH/ 35 Date 1/1/24

**The Principal**
**Dayanand Medical College & Hospital**
**Ludhiana**

**Subject:: Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 25.09.2023 has approved the plan of thesis of **Purnima**, a student of your college for **MD Pathology** along with the names of Supervisor/Co-Supervisor as under:-

**Topic Plan of Thesis**
Histopathological spectrum of hyperpigmented skin lesions .

| **Supervisor** | **Co-Supervisor:-** | **Co-Supervisor:** |
|---|---|---|
| Dr. Bhavna Malhotra | Dr. Vikram Narang | Dr. Sukhjot Kaur |
| Professor | Assoc. Prof., | Professor |
| Dept. of Pathology | Dept. of Pathology | Dept. of Dermatology |

The student may submit the thesis in triplicate alongwith the plan in triplicate also, to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She may be directed to attach a photocopy of this letter with the forwarding letter while submitting thesis in the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic, if any.

Controller of Examinations

*Note :*

1. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*

# HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED SKIN LESIONS

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED IN

PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

MD

In

PATHOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES

FARIDKOT

AUGUST, 2022

DR. PURNIMA

DEPARTMENT OF PATHOLOGY

DAYANAND MEDICAL COLLEGE AND HOSPITAL

LUDHIANA

# ABSTRACT OF PLAN OF THESIS

| | |
|---|---|
| Title | HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED SKIN LESIONS |
| For the degree of | MD (PATHOLOGY) |
| Name of the candidate | DR. PURNIMA |
| Supervisor | Dr. Bhavna Malhotra, Professor, Department of Pathology , DMCH , Ludhiana |
| Co-supervisor(s) | Dr. Vikram Narang, Associate Professor, Department of Pathology, DMCH, Ludhiana<br><br>Dr. Sukhjot Kaur, Professor, Department of Dermatology Venerology and Leprosy, DMCH, Ludhiana |
| Institution | Dayanand Medical College and Hospital, Ludhiana |
| University | Baba Farid University of Health Sciences, Faridkot |

**Introduction**: All the hyperpigmented skin lesion biopsies will be stained with H&E stain after fixation in 10% formalin and analysed in detail. Special stains and IHC will also be performed wherever required.

**Aim of Study**: To analyse the histological spectrum of hyperpigmented skin lesions.

**Materials & methods**: Data for the prospective study will be obtained from all the skin biopsies of hyperpigmented skin lesions received in the Department of Pathology, DMCH, over a period of 1.5 years starting from 15 September 2022 to 8 March 2024. All the slides will be stained with H&E stain after fixation in 10% formalin and analysed in detail. Special stains and IHC will also be performed wherever required. These findings will be correlated with clinical profiles of the patients.

**Clinical Significance**: To obtain reasonable good database for a more accurate assessment and diagnosis of hyperpigmented skin lesions.

**Keywords**: hyperpigmentation, benign, malignant, lichen planus, melasma

| (Signature) | (Signature) | (Signature) | (Signature) | (Signature) |
|---|---|---|---|---|
| Candidate | Co-supervisor | Co-supervisor | Supervisor | Principal |

## CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED SKIN LESIONS"** exist in the Department of Pathology, DMC&H, Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate herself under our supervision and guidance.

**Supervisor**

Bhavna

**Dr Bhavna Malhotra**
Professor
Department of Pathology
DMCH, Ludhiana

**Co-Supervisor's**

**Dr Vikram Narang**
Associate Professor
Department of Pathology
DMCH, Ludhiana

Sukhjot Kaur

**Dr. Sukhjot Kaur**
Professor
Department of Dermatology
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**Head of the Department**

Harpreet

**Dr. Harpreet Kaur**
Professor & Head
Department of Pathology
DMCH, Ludhiana

Place : Ludhiana

Date : _____

# APPROVAL PROFORMA
## BY RESEARCH & ETHICAL COMMITTEE
## DAYANAND MEDICAL COLLEGE & HOSPITAL, LUDHIANA

| | |
|---|---|
| **Name of candidate** | Dr. Purnima |
| **Department** | Pathology |
| **Topic of Thesis** | HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED SKIN LESIONS |
| **Likely date of appearing in PG examination** | May/ June 2024 |
| **Date of enrollment** | 7th March, 2020 |
| **Name of Head of Department** | Dr. Harpreet Kaur |
| **1) Supervisor**<br><br>**2) Co-Supervisor(s)** | Dr. Bhavna Malhotra<br><br>Dr. Vikram Narang<br>Dr. Sukhjot Kaur |
| **Signature of Members of Research Committee with Stamp** | **Signature of Members of Ethics Committee with Stamp** |
| Dr. Harpreet Kaur<br>(Professor & Head, Pathology) | Mr. R K Sharma<br>(Legal Expert) |
| Dr. Jaspal Singh<br>(Professor & Head, Surgery) | Dr. Ramit Mahajan<br>(Associate Professor, Gastroenterology) |
| Dr. Dinesh Gupta<br>(Professor & Head, Medicine) | Dr. Anu Sharma<br>(Professor, Anatomy) |
| Dr. Sandeep Kaushal<br>(Dean Academics & Professor & Head, Pharmacology) | Dr. Sandeep Kaushal<br>(Dean Academics & Professor & Head, Pharmacology) |
| Dr. Gurpreet S Wander<br>(Vice Principal) | Dr. Gurpreet S Wander<br>(Vice Principal & Professor & Head, Cardiology) |
| | Dr. Gagandeep Singh<br>(Secretary, Ethics Committee Professor & Head, Neurology) |

**Approved: Yes / ~~No~~**

**Dr. Sandeep Puri**
**Chairperson**
**Research Committee**

Chairperson
Research Committee

**Approved: Yes / ~~No~~**

**Prof. Arvind Malhotra**
**Chairperson**
**Ethics Committee**

Chairperson
Ethics Committee

# BABA FARID UNIVERSITY OF HEALTH SCIENCES, FARIDKOT

## PROFORMA FOR REGISTRATION OF SUBJECT FOR DISSERTATION

| | | |
|---|---|---|
| 1 | NAME OF THE CANDIDATE AND ADDRESS | Dr Purnima<br><br>VPO Jandwala Bishnoiyan, Tehsil Mandi Dabwali, Distt. Sirsa, Haryana |
| 2 | NAME OF THE INSTITUTE | Dayanand Medical College and Hospital, Ludhiana |
| 3 | COURSE OF STUDY AND SUBJECT | MD Pathology |
| 4 | DATE OF ADMISSION OF COURSE | 07-03-2022 |
| 5 | TITLE OF THE TOPIC | HISTOPATHOLOGICAL SPECTRUM OF HYPERPIGMENTED SKIN LESIONS |

## 6. BRIEF RESUME OF THE INTENDED WORK

### 6.1 NEED FOR THE STUDY

The skin is the largest organ of the body, accounting for about 15% of total body weight in humans. The wide range of different colours and gradations of the human skin is due to the presence of melanin.

Pigmentary problems are considered one of the major factor for dermatological consultation. In India , they have a great psychological impact on the quality of life. The amount of melanin present in the human skin can lead to hypopigmented and hyperpigmented skin lesions.[1]

Hyperpigmentary skin disorders may be defined as increased pigmentation of the skin and/or mucous membranes. Hyperpigmentation may be caused by abnormality in epidermis or dermis. Hyperpigmentary skin disorders may be broadly classified into two groups, epidermal which is characterized by 'Brown hyperpigmentation' and dermal which is known as 'blue hyperpigmentation' or 'Ceruloderma.

Pigmented skin lesions comprise a large component of most histopathologists' workload, and there is a wide spectrum of histological appearances ranging from non- neoplastic conditions like nevi to neoplastic conditions like malignant melanoma.

Disorders of pigmentation can result from-

- migration abnormalities of melanocytes from neural crest to the skin during embryogenesis
- impairment of melanosome transfer to keratinocytes
- alteration in melanin synthesis.

Immunologic or toxic mediated destruction of melanocytes is another cause of pigmentaion disorder. Various common drugs like antibiotics and diuretics also stimulate melanogenesis and increase melanin

content in large melanocytes. Heavy metals also elicit hyperpigmentation by binding and thereby inactivating sulfhydryl compounds in the skin that normally inhibit tyrosinase activity. Removal of this inhibition results in increased melanogenesis.[2]

Hyperpigmentation is not just a cosmetic deformity causing psychological upset due to disfigurement, but can also reflect underlying organ dysfunction.

When the patient seeks medical attention for skin lesions, the physician requires an accurate diagnosis in order to institute proper treatment. However, the clinical diagnosis of skin diseases may be challenging, as the clinical information and appearance of skin lesions invariably overlap. Correct diagnosis often lacks without histopathological examination of skin biopsies.[3]

## 6.2 REVIEW OF LITERATURE

Our planet earth has variegated collection of people with different skin colours. More than a century ago, the skin was considered as a protective covering for more delicate and functionally sophisticated internal viscera. Then, for the century that followed, the skin was considered primarily a passive protective barrier to both fluid loss and mechanical injury. In recent years, there have been various researches related to the melanocytes, clinical aspects and treatment of pigmentary disorders.

The epidemiology of pigmentary disorders varies in different areas of the world.[2]

NORMAL STRUCTURE OF SKIN

Skin is composed of three layers: (a) epidermis (b) dermis, and (c) the subcutaneous adipose tissue. Functions of the skin are extremely diverse.

• It serves as a mechanical barrier against external physical, chemical, and biologic noxious substances and as an immunologic organ.

• It participates in body temperature and electrolyte regulation.

• It is an important organ of sensuality and psychological well-being. In addition, it is a vehicle that expresses not only primary diseases of the skin, but also diseases of the internal organs.

EMBRYOLOGY

• The epidermis begins as a single layer of ectodermal cells. By 5 weeks of gestational age, this differentiates at least focally into two layers—the basal layer or stratum germinativum and the overlying periderm.

• By 10 weeks, an intervening layer—the stratum intermedium develops.

• By 19 weeks, there are several layers of intermediate cells, and the periderm begins to flatten.

Keratinization is well developed by 23 weeks within the stratum intermedium in association with small keratohyaline granules. At this juncture, most of the periderm cells have shed, and the keratinizing cells that remain beneath represent the newly formed stratum corneum.

KERATINOCYTES

The keratinocytes of the epidermis are stratified into four orderly layers from bottom to top:

(a) the basal layer (stratum basale, germinativum),

(b) the squamous layer (prickle cell layer or stratum spinosum),

(c) the granular layer (stratum granulosum),

(d) the cornified or horny layer (stratum corneum) .

Melanocytes

Melanocytes are dendritic cells that derive from the neural crest. During migration from the neural crest, melanocytes may localize in other epithelia. In the epidermis, the melanocytes are localized in the basal layer, and their dendritic processes extend in all directions. In H&E preparations, melanocytes are composed of elongated or ovoid nuclei surrounded by a clear space. They are usually smaller than the neighboring basal keratinocytes. Melanocytes do not contain tono filaments and do not attach to basal cells with desmosomes. Laminin-5, a component of anchoring filaments which extend from plasma membrane of the melanocytes to the basal lamina, may be a ligand for melanocyte attachment to the basement membrane in vivo. In addition, melanocytes that are close to the basal lamina have structures resembling hemidesmosomes of basal keratinocytes .

**CLASSIFICATION OF HYPERPIGMENTED SKIN LESIONS[3]**

1. Congenital
2. Acquired
   (i) Acquired localised

Circumscribed lesions :

-Ephelids

-Lentiginosis

-Melasma

-Exogenous ochronosis

-Fixed drug eruption

-Frictional melanosis

-Reihl's melanosis

-Post inflammatory hyperpigmentation - Eczema

Lichen planus

Lichenoid eruption

Amyloidosis

Morphea

Erythema dyschromicum perstans

Pemphigus vulgaris

Disseminated lesions :

-Endogenous ochronosis

-Mastocytosis

-Phytophotodermatosis

-Idiopathic eruptive macular pigmentation

-Lichen planus pigmentosus

(ii) Acquired diffuse

- Endocrinopathies
- Nutritional conditions
- Metabolic conditions
- Neoplastic / tumoral
- Physical agents
- Toxins / drug induced
- Progressive systemic sclerosis

**EPHELIDS**

Ephelids (freckles) are small pigmented macules occuring on face and less commonly on other sun exposed areas. Freckles are irregular

small macules of about 1-2 mm in size, commonly seen over nose and malar area.

Histological features include hypermelanization of basal epidermis and no increase in number of melanocyte.[4]

**MELASMA**

Melasma is a common acquired condition of symmetric hyperpigmentation, typically occuring on the face, with higher prevalence in females and dark skin tones. Multiple etiological factors including light exposure, hormonal influences and family history have been implicated in its pathogenesis. Histologically, melasma can display increased epidermal and /or dermal pigmentation, enlarged melanocytes, increased melanosomes, dermal blood vessels and occasional perivascular lymphohistiocytic infiltrates.

Various topical, oral and procedural therapies have been successfully used to treat melasma.[4]

Mohammad Ali *et al* did a retrospective study on patients presenting with complaints of skin hyperpigmentation from January to December 2016. Out of 7124 patients, melasma was considered the most common cause of skin hyperpigmentation seen in 33.63% patients followed by post inflammatory hyperpigmentation seen in 12.52% patients. Thus concluding melasma to be the leading cause of hyperpigmentation.[5]

**FIXED DRUG ERUPTION**

It presents as recurrent plaques at fixed location such as lips, genital areass and acral areas after every exposure to offending drug. These plaques are non itchy and resolve spontaneously with post-inflammatory hyperpigmentation.

Histologically, necrotic keratinocytes and lymphocytic infiltrates are seen that obscures dermaoepidermal junction. Pigment incontinence is a prominent feature. Dermal melanophages may be the only tell- tale sign of a resolved lesions of FDE.[4]

## OCHRONOSIS

Exogenous ochronosis is a blue black dermal discolouration secondary to use of bleaching creams which contain hydroquinone. Clinically, bony prominences are involved where pigmentation occur and on other areas like forehead, temple, nose, lower jaw and side of neck.

Histopathology shows swollen and basophilic collagen fibres. Older lesions show yellow brown ochronotic pigment (homogentisic acid) coating collagen fibres appearance as banana shaped bodies. The pigment does not stain with Masson fontana or perl's stain. Histopathological features are common to both endogenous and exogenous ochronosis.[4]

## POIKILODERMA OF CIVATTE

Poikiloderma of civatte is most commonly seen in middle aged, postmenopausal caucasian women, but it has also been seen in individuals with skin of colour. Lesions on the skin involve sun exposed areas of face, neck, and V of chest. Clinically it is seen as reticulate spots of hyperpigmentaion.

Histologically it is characterised by telengectasia, fibrosis and melanophages in the papillary dermis with prominent solar elastosis. Accumulated elastotic material can be better visualized with an Orcein stain.[4]

## CUTANEOUS AMYLOIDOSIS

Amyloidois is a condition characterized by extracellular deposition of fibrillar proteins which is responsible for tissue damage and functional compromise. Primary localised cutaneous amyloidosis is the deposition of amyloid in an apparently normal skin.

Histopathologically, amyloid appear as eosinophilic material with H&E stain. Congo red staining with apple green birefringence under polarized microscopy confirms the diagnosis.[6]

Venugopal SB *et al* in their study showed that majority of the lesions of cutaneous amyloidosis were pruritic with involovement of pretibial region predominantly. Microscopically, hyperkeratosis, amyloid

deposits in the papillary dermis and perivascular inflammation were the most consistent findings in all the cases.[6]

**RIEHL'S MELANOSIS**

A pigmented contact dermatitis in response to cosmetics presents itself as grey brown pigmentation. It is predominantly seen in middle aged women. The condition represents a Type IV hypersenstivity reaction and leads to basal cell vacuolization, pigment incontinence and a perivascular or a band like dermal infiltrates in the early lesion. The pattern can be diffused, patchy or reticular with satellite, perifollicular pigmented macules.[7]

**IDIOPATHIC ERUPTIVE MACULAR PIGMENTATION**

It presents with asymptomatic brown macular lesions over the trunk area, proximal extremities and neck. This condition is most commonly seen in children and adolescents.

Histology shows pigmentation of the epidermal basal layer without degeneration and the lesions may be resolved in a few months.[7]

**SEBORRHEIC KERATOSIS**

Seborrheic keratosis are one of the commonest benign and pigmented epidermal tumours. They usually develop after the age of 50 years without any sex predeliction.

Heredity, sunlight and human papilloma virus (HPV) have been suggested as risk factors.

These lesions are slightly raised, tan to brown or black papules. Sun exposed skin is commonly affected, but lesions may be present on any site of the skin except for palms and soles. They often have a stuck on appearance and may be easily removed.

Seborrheic keratoses are well defined proliferations of epidermal keratinocytes which may be endophytic, exophytic or flat. Seborrheic keratoses show a variety of histologic appearances.

All types of seborrheic keratosis have in common hyperkeratosis, acanthosis, and papillomatosis. Two types of cells are usually seen in

the acanthotic epidermis: squamous cells and basaloid cells. The former have the appearance of squamous cells normally found in the epidermis; the basaloid cells are small and uniform in appearance and have a relatively large nucleus.

Follow-up for patients with multiple seborrheic keratoses is important because malignant tumors can develop elsewhere on the body (or rarely within a seborrheic keratosis).Treatment is usually not required unless the growths become irritated or are cosmetically displeasing. If treatment is needed, growths may be surgically removed or removed by cryotherapy (freezing).

**LICHENOID ERUPTION**

Lichenoid tissue reaction (LTR) or interface dermatitis (IFD) is some of the commonly encountered clinical and histological presentations in dermatology and pathology.

The term interface dermatitis refers to the finding in a skin biopsy of an inflammatory infiltrate that obscures the dermo-epidermal junction.

Histopathologically, differentiating features of lichenoid eruptions include focal parakeratosis and mild basal vacuolar changes with a few eosionophils and plasma cells.[8]

**LICHEN PLANUS**

Lichen planus is the most common variant of interface dermatitis (lichenoid tissue reaction) which involves skin, mucous membranes, hair follicles and nails.

It is an eruption of unknown etiology, displaying violaceous, flat topped papules, which are usually pruritic. A network of fine white lines (Wickham’s striae) may be seen on the surface of papules. There is a predilection for the flexor surface of the wrists, trunk, thighs, and genitalia.

LP is characterized by lichenoid interface dermatitis.

The classic histopathological features include a dense, continuous, and band-like lymphohistiocytic infltrate at the dermal-epidermal junction and in the upper dermis.

Epidermal changes in LP lesions include irregular epidermal hyperplasia with a jagged “sawtooth” appearance, compact hyperkeratosis or orthokeratosis, foci of wedge-shaped hypergranulosis, basilar vacuolar degeneration, slight spongiosis in the spinous layer, and squamatization. The dermal papillae between the elongated rete ridges are frequently dome shaped. Necrotic keratinocytes can be observed in the basal layer of the epidermis and at the dermalepidermal junction. Eosinophilic remnants of anucleate apoptotic basal cells may also be found in the dermis and are referred to as “colloid or civatte bodies”. Wickham striae are usually seen in the areas of hypergranulosis .Vacuolar degeneration at the basal layer may be noted leading to focal subepidermal clefts (Max Joseph spaces).

Mruthunjayappa *et al* proved in their study that the most common biopsy confirmed hyperpigmented lesions included classical lichen planus and its variants (55%). Histopathologically, most of the lesions showed the presence of interface dermatitis.[8]

Gourohi F *et al* showed that vulvar lichen planus may interfere with sexual intercourse and disturb the psychological and physical well being of the individual. There is also increased prevalence of HCV and HPV infection in such patients.[9]

**PRURIGO NODULARIS**

Prurigo nodularis is a chronic dermatitis characterized by discrete, raised, firm hyperkeratotic papulonodules of 5 to 12 mm size and are intensely pruritic. They have a predeliction for the extensor surfaces of extremities.

Prurigo nodularis is a condition of unknown etiology. Many conditions have been reported to induce prurigo nodularis, from metabolic or systemic diseases to psychiatric conditions.

Chronic mechanical trauma to the skin like scratching results in plaques or nodular lichenification and hyperkeratosis, along with pigmentary changes and increased number of dermal nerve fibers.

On histopathology, marked hyperkeratosis and irregular acanthosis is seen. Hypertrophy and proliferation of dermal nerves are the characteristic neurological change seen in such patients.[1]

**MORPHEA**

Morphea is characterized by purplish or mauve color with lilac border. On Fontana Masson stained sections there is increased melanocytic activity in the form of prominent melanocytes in the basal cell layer. There is no basal cell degeneration and melanin incontinence.

Histopatholgy of morphea is termed early inflammatory, intermediate and late sclerotic stages. Early lesion shows interstitial lymphoplasmacytic infiltrates with or without eosinophils among slightly thickened collagen bundles. Late sclerotic stage shows more thickened collagen bundles with almost normal epidermis and complete disappearance of inflammatory infiltrate.[1]

**HYPERPIGMENTATION DUE TO PHYSICAL AGENTS**

Hyperpigmentation due to physical agents may be due to exposure of skin to ultraviolet radiation; UVA radiation being most common to to cause tanning. There are other causes such as ionizing radiation, thermal radiation and trauma.[7]

**NUTRITIONAL DEFICIENCY DISORDERS**

Nutritional deficiency disorders are known to cause a variety of cutaneous manifestations. Some of them are malabsorption syndrome, vagabond's disease and most commonly involved are the vitamin deficiencies (vitamin A, vitamn B3, vitamin B12, folic acid, vitamin C).[7]

**ENDOCRINOPATHIES**

They can lead to a variety of cutaneous manifestations, including hyperpigmentation of skin which often serves as an indicator of underlying endocrine disturbance. These changes can be oral contraceptives induced, pregnancy induced or various endocrinopathies.[7]

## POST INFLAMMATORY HYPERPIGMENTATION

Post inflammatory hyperpigmentation is manifested due to sun insult particularly in people with dark skin, such insults are acne lesions, ingrown hairs, scratches and insect bites. It presents as discrete, hyperpigmented macules with hazy feathered margins, which may involve epidermis or dermis. Histologically the sun exposed skin has elevated inflammatory cell count. Epidermal postinflammatory hyperpigmentation involves increased melanin pigment in the basal cell layer of the epidermis.

Dermal postinflammatory hyperpigmentation involves the upper dermis, with pigment incontinence due to increased numbers of melanophages in the papillary dermis.

Krishma Goyal *et al* in their study showed that two major causes of pigmentary disorders were post inflammatory hyperpigmentation and lichen planus pigmentosis.[1]

Histopathologically, all the cases showed perivascular lymphocytic inflammatory cell infiltrate with majority of them showing pigment incontinence. Other features seen in PIH include increased basal cell pigmentation, hyperkeratosis and basal cell degeneration.[4]

## MELANOCYTIC LESIONS

Melanocytic lesions exhibit great morphological diversity in their architecture and cytomorphological appearance of their composite cells.

Some factors have emerged from studies that can predict the development of melanocytic nevi, the most important being sun exposure in childhood. The emergence of nevi is under strong genetic control, whereas environmental exposures affect the mean number of nevi.

Melanocytic proliferations are composed of one or more of three related types of cells: melanocytes, nevus cells, and melanoma cells, each of which may be located in the epidermis, the dermis, or occasionally also in the subcutis. Localized proliferation of melanocytes may occur, giving rise to benign or malignant melanocytic neoplasms,

characterized by melanocytes that lie in contiguity with one another to form a tumor.

Benign tumors of melanocytes are generally called melanocytic nevi, whereas malignant tumors are called malignant melanomas, and the cells of these lesions are called nevus cells and melanoma cells, respectively.

**Melanocytic nevi**

Melanocytic nevus is generally considered to be a benign neoplastic proliferation of melanocytes. Melanocytic nevi are only rarely present at birth. Most nevi appear in adolescence and early adulthood.

There are transitional stages in the life cycle of nevi,which are believed to start out as junctional nevi, then become compound nevi and after having become intradermal nevi, undergo involution.

Junctional Nevi

It is seen as a brown or black macule present anywhere in the body mostly seen during childhood or early adolescence.

It is composed of nevus cells that lie in well circumscribed nests either entirely within the lower epidermis or bulging downward into the dermis but still in contact with the epidermis. The nevus cells in these nests generally have a regular, cuboidal appearance, although they are occasionally spindle-shape.Varying amounts of melanin granules are seen in the nevus cells.

Compound Nevi

They occur more commonly in children and adolescents. Clinically, a compound nevus is a pigmented papule or a plaque.

A compound nevus possesses features of both a junctional and an intradermal nevus.

Nevus cells in the upper, middle, and lower dermis may present characteristic morphologic variations called types A, B, and C, respectively.

Usually, the type A nevus cells in the upper dermis are round to cuboidal and show abundant cytoplasm containing varying amounts of melanin granules. Melanophages are occasionally seen in the surrounding stroma.

The cells in the mid-dermis usually are type B cells. They are smaller than the type A cells, display less cytoplasm and less melanin, and generally lie in well-defined aggregates or cords.

Type C nevus cells in the lower dermis tend to resemble fibroblasts or Schwann cells, because they are usually elongated and possess a spindle-shaped nucleus. They often lie in strands and only rarely contain melanin.

Intradermal Nevus

It is the most common type of melanocytic nevi and vast majority are found in adults.

Intradermal nevi show essentially no junctional activity. Nevus cells are confined to the dermis where they are arranged in nests and cords. Multinucleate cells may be seen. In the deeper parts of the lesion nevus cells may assume a neuroid appearance.

For the treatment of a benign melanocytic nevus, cosmetic improvement becomes the main purpose. Many surgical and non-surgical treatment modalities have been reported. Large congenital melanocytic nevi carry a high risk of malignancy before the age of ten years. Therefore, the current recommended treatment for these lesions is early and complete excision followed by reconstruction. Recently, the progress of laser treatment has been remarkable as new therapy.

**Malignant melanoma**

The term “black cancer” was first used to describe cutaneous malignant melanoma by Hippocrates in the fifth century BC. In 1806, Rene Laennec provided the first description of melanoma as a disease entity and also marks the first published use of the word melanoma.

The peak incidence is around the sixth decade of life.

Most commonly they are found in the head and neck area and lower extremity, the latter being particularly common in females.

It is now widely accepted that the major environmental risk factor for the development of primary cutaneous melanoma is Ultraviolet Radiations, which can be subdivided into ultraviolet A (UVA), ultraviolet B(UVB),and Ultraviolet C (UVC).

Environmental exposure (UV light) and genetic susceptibility (CDKN2A, CDK4, MC1R, BRAF, p16/ARF) leads to the accumulation of genetic mutations in melanocytes that activate oncogenes, inactivate tumour suppressor genes and impair DNA repair mechanisms. This leads to melanocyte proliferation,blood vessel growth,tumour invasion, suppression of immune response and ultimately metastasis.

Histology shows the presence of intra and extracellular melanin in the dermis along with very prominent perivascular distribution.[7]

Melanoma may occur de novo, that is without a precursor lesion, or may develop within a pre-existing benign melanocytic nevus. All types of melanocytic nevi can give rise to melanoma, but some are more frequently involved such as congenital melanocytic nevi.

Tumor thickness and lymph node metastasis are considered to be important prognostic factors of malignant melanoma.[10]

Nam KM *et al* in their study showed that most common site for the primary skin tumors include lower extremities followed by feet, upper extremities, trunk and head and neck. Among the histopathological subtypes, the most common to be seen was acral lentiginous melanoma followed by nodular melanoma, superficial spreading melanoma and lentigo maligna melanoma.[10]

**NEVUS OF OTA AND ITO**

Nevus of ota and ito are two benign melanocytic conditions which differ only on the basis of site of involvement and distribution.

Nevus of ota involves the trigeminal nerve distribution, mainly the first and second division of trigeminal nerve namely ophthalmic V1 and maxillary V2. Hyperpigmentation of the eye is also seen which gives its other name as ocular dermal melaonsis.

Nevus of ito involves the distribution area of lateral cutaneous brachial nerves of shoulder and posterior supraclavicular nerves. Ocular complications are not seen with nevus of ito.

Both of these conditions show similar pathophysiology of increased melanocytes in the dermis.[11]

Agarwal P *et al* showed in their study that both these conditions have no definite diagnostic test. They can be diagnosed mainly by clinical examination and history. Skin biopsy becomes mandatory if the overlying skin becomes ulcerated or there is variation in pigmentation.[11]

**PROGRESSIVE SYSTEMIC SCLEROSIS**

Systemic sclerosis is a chronic multisystem connective tissue disorder characterised by multiple manifestations in the body.

It is characterised by microangiopathy leading to inflammation and fibrosis of the skin and underlying organs. The exact mechanism of diffuse pigmentation of the body is not known.

Viswanath V *et al* in their study gave the reason of hyperpigmentation of the body to be melanocytic potential of endothelin-1 (ET-1) which is produced in keratinocytes and stimulates melanogenesis by multiplication of melanocytes and melanin synthesis.[12]

**URTICARIA PIGMENTOSA**

It is also known as mastocytosis and is characterised by mast cell accumulation in the skin, bone marrow, GIT, liver, spleen and lymphatic tissues.

Diffuse cutaneous involvement is seen in the advanced stages of mastocytosis. Is is considered to be benign, self resolving condition which remits in adolescence.

Diagnosis of urticaria pigmentosa can be made clinically but a definite diagnosis requires a skin biopsy. The hallmark finding of urticaria pigmentosa is mast cell accumulation in the dermis.

Macri *et al* in their study showed that biopsy specimens from normal skin of the patient of mastocytosis did not show an increased number

of mast cells in the dermis. In such patients, a bone marrow biopsy or biopsy from GIT might be needed.[13]

Jaykaer SS *et al* included 85 cases of histologically diagnosed hyperpigmneted skin lesions. Clinical features like age, sex, type of pigmentation and location were also included. Out of these 85 cases, 12.94% were of classical lichen planus with the majority of the cases in the age group of 21-30 years and showed female preponderance with extremities as the most common site of ditribution. Thus concluding that hyperpigmented skin leisons are the most common in females with lichen planus being the most common diagnosis.[14]

A study was done in 2020 by Dr Abhishek Singh *et al* at the department of pathology at a tertiary care hospital and 75 cases of pigmented skin lesions were reviewed on H&E stain. Out of the 75 cases, 23 were melanocytic lesions and 52 were non melanocytic. Overall, benign melanocytic nevi were the most common lesions. The most common site involved was head and neck as observed in this study. Thus concluding that pigmented skin lesions are common presenting problem and majority of them are benign lesions.[15]

Dr. PRD Ganesh Basina *et al* did a prospective study in the department of pathology of Rangaraya Medical College, Kakinada from June 2015 to June 2016. 60 cases of hyperpigmented skin lesions were evaluated. The age distribution of the cases were from 5 to 75 years. The male to female ratio came out to be 1.1:14. Out of the total 60 cases, the total number of non melanocytic lesions constitute 93.33% where as the melanocytic lesions were only 6.67%. The study concluded that inflammatory lesions were the most common constituting 66.66% and lichen planus and its variants being the most common of it (20%). In their study, it was seen that out of 60 pigmented cutaneous and mucosal lesions, nonmelanocytic inflammatory, benign and malignant lesions constitute 93.3% and melanocytic benign and malignant lesions constitute 6.6%. Thus concluding that

hyperpigmented leions of the skin and mucous membranes range from inflammatory to malignant. Lichen planus and its variants were the most common pigmented lesions in this study.[16]

There is another study done by Dr Sneha Ravindran *et al* in the year 2020 on hyperpigmented skin lesions in which a total of 100 patients with hyperpigmented punch skin biopsy lesions in all age groups were included. The maximum number of cases 45% were of classical lichen planus and its variants, followed by 12% of eczematoid dermatitis, 8% of PMLE and 7% of DLE. Thus concluding that lichen planus an dits variants are the most common hyperpigmentary skin lesions found in the study done.[17]

There was another study done by Ahmed SA *et al* in 2021 in which retrospective study was done for last 3 years from January 2017 to December 2019 in the pathology department of tertiary care centre on all skin biopsy tissues which came as pigmented lesion. Out of 58 cases of pigmented skin lesions, 5 biopsies had inadequate material and 5 gave non specific findings. Of the remaining cases, 15 were benign and 33 were malignant. Only 18 of these cases were of melanocytic origin with 8 cases of malignant melanoma and 10 cases of nevi. This study showed a positive correlation (54%) between clinical and histopathological diagnosis. Thus concluding that a good clinical correlation and biopsy with histopathological diagnosis is necessary for the accurate diagnosis and definite treatment of patients with pigmented skin lesions.[18]

## 6.3 AIMS AND OBJECTIVES OF THE STUDY

1. To analyse the histological spectrum of hyperpigmented skin lesions.

## 7. MATERIALS AND METHODS

### 7.1 Source of Data

Data for the prospective study will be obtained from all the skin biopsies of hyperpigmented skin lesions received in the Department of Pathology, DMCH, over a period of 1.5 years starting from 15 September 2022 to 8 March 2024

### 7.2 METHOD OF COLLECTION OF DATA

**INCLUSION CRITERIA**

Skin biopsies of all the hyperpigmented lesions of the skin received in Department of Pathology, DMCH

**EXCLUSION CRITERIA-**

- All other skin biopsies which are not hyperpigmented.

**Methodology:**

All the slides will be stained with H&E stain after fixation in 10% formalin and analysed in detail. Special stains and IHC will also be performed wherever required. These findings will be correlated with clinical profiles of the patients. Pictures of all the hyperpigmented skin lesions at the time of presentation will be attached wherever possible.

**OUTCOME**

All the H&E stained skin biopsy slides will be analysed in this study. The clinical diagnosis will be correlated with the histopathological diagnosis.

**STATISTICAL ANALYSIS**

Data will be described in terms of range; mean ± standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21 version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

**7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.**

No.

**7.4 HAS ETHICAL CLEARANCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?**

Yes, Attached herewith.

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# PROFORMA

SR No: Age and sex:
Name / Patients initials: MRD No:
Histopath No: IHC No:
Date of receiving the biopsy: Date of reporting the biopsy:
Chief complaints:
Duration:

**History of present illness**:

Mode of onset and symptoms : sudden / gradual

Progression of disease : present/ absent

Remission and relapse : present / absent

Aggravating factors : Sun exposure- present/ absent

Any drug reactions- present/absent

Hormonal imbalance- present/ absent

Skin injuries - present/absent

H/O any drug intake- present/absent

H/O hair dye use- present/absent

Use of cosmetics- present/absent

**Past history** :

Similar episodes : present/absent

Other diseases:

**Investigations** : Hb TLC DLC

- Any other positive finding in any system of the body(if any)/ other investigations:

**Signature of Candidate** **Signature of Supervisor**

**Examination of the cutaneous lesion :**

- Morphology of the lesion : Macular / papular / plaques / nodular
- Number of lesions :
- Site of lesions:
- Symmetry : unilateral /bilateral
- Size of lesions:
- Distribution of lesions : extensors/ flexors/ any other if specified
- Colour of skin lesion:
- Any other cutaneous finding:

**Clinical diagnosis** :

| **NON MALIGNANT LESIONS** | | **MALIGNANT CELLS** | |
|---|---|---|---|
| LICHEN PLANUS | | BASAL CELL CARCINOMA | |
| LICHEN PLANUS PIGMENTOSIS | | MELANOMA | |
| PIGMENTED COSMETIC DERMATITIS | | ANY OTHER | |
| FIXED DRUG ERUPTION | | | |
| CUTANEOUS AMYLOIDOSIS | | | |
| POST INFLAMMATORY PIGMENTATION | | | |
| SEBORRHEIC DERMATITIS | | | |
| LICHENOID DRUG RASH | | | |
| MELANOCYTIC NEVUS | | | |
| DRUG INDUCED PIGMENTATION | | | |
| ANY OTHER | | | |

**Site of biopsy** :

**Histopathological examination** :

**Epidermis** : Hyperkeratosis- present/absent

Parakeratosis- present/absent

Acanthosis- present / absent

Hypergranulosis- present/absent

Spongiosis- Present/absent

Basal cell degeneration- present/absent

Inflammatory infiltrate- present/absent

Thinning of epidermis- present/ absent

Civatte bodies- present/ absent

Max Joseph's space - present/ absent

Saw toothed rete ridges - present/ absent

Follicular plugging - present/ absent

Thickening of basement membrane- present/ absent

Pigment in basal layer- present/ absent

Horn cysts - present/ absent

Squamous eddies - present/ absent

Necrotic keratinocytes- present/absent

Ulceration - present/ absent

Dysplasia - present/ absent

Malignant cells - present/ absent

**Dermis** : Infiltration- present/absent

Location- superficial dermis/ deep dermis

Type of cells- Lymphocytes/ plasma cells/ mast cells/ neutrophils/ eosinophils

Band-like inflammatory infiltrate - present/ absent

Perivascular infiltrate - acute/ chronic

Periadnexal infiltrate - acute/ chronic

Epitheloid cell granuloma - present/ absent

Pigment incontinence - present/absent

Basement membrane thickening - present / absent

Increased collagenisation - present/ absent

**Signature of Candidate** **Signature of Supervisor**

Fibrosis in Papillary dermis - present/ absent

Amyloid deposits in Papillary dermis - present/ absent

**Nevus cells** : present/ absent

- Location – Epidermal/ dermal

- Maturation : present/ absent

- Necrosis : present/ absent

- Mitosis : present/ absent

- Junctional activity : present/ absent

**Melanoma cells** : present/ absent

- Necrosis : present/ absent

- Vertical spread : present/ absent

- Horizontal spread : present/ absent

Any other malignant cells :

**Subcutaneous tissue**: present/ absent

- Inflammatory infiltrate: present/ absent

- Type of cells: Lymphocytes/plasma cells/ Mast cells/ neutrophils/ eosinophils/ epithelioid cells/ granulomas

**Special stains** :

**IHC findings if any** :

**Final diagnosis** :

**Signature of Candidate** **Signature of Supervisor**